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CARDIAC PAIN.[1] [2]

By W. E. FISHER, M.D., Ch.M. (Sydney), M.R.C.P. (London),

Vice-Master and Tutor in Medicine, Wesley College, University of Sydney; Honorary Relieving Assistant Physician, Sydney Hospital; Honorary Assistant Physician, Royal South Sydney Hospital and Eastern Suburbs Hospital.

(From the Department of Medicine, University of Sydney.)

THE investigations on which this paper is based were undertaken because I realized that the current concept of *angina pectoris* failed to satisfy everyday clinical requirements. In lodge practice and out-patient work many patients were seen who complained of pain in the front of the chest on the left side; undergraduate days had left the impression of *angina pectoris* as a paroxysm, usually of characteristic radiation, and frequently associated with *angor animi*; post-graduate work had added, as a refinement, a clear-cut idea of cardiac infarction; but these patients presented various pictures. Their pain was sometimes severe, sometimes retrosternal, sometimes of classical radiation, sometimes brought on by cold, emotion or effort; on the other hand, it was often submammary rather than retrosternal, of long duration, stopped and started gradually, and was rarely accompanied by *angor animi*. In other words, its site varied, *angor animi* was the exception rather than the rule, and it was not paroxysmal. It did not accord with *angina pectoris* as described by Heberden,[3] nor did it fulfil the criteria recently established for cardiac infarction. Take for example

[1] The work on which this thesis is based was done under a tenure of the Marion Clare Reddall Scholarship, 1931-1932.

[2] Submitted as a thesis for the degree of Doctor of Medicine in the University of Sydney.

the case of Mrs. C.E. (Case X), showing pain, definitely severe, and associated with organic cardiovascular disease, but of long duration, not associated with *angor animi*, and in many sites.

This woman, aged fifty-eight years, has had for ten years a systolic pressure ranging from 220 to 260 millimetres of mercury. She is troubled at times by palpitation and breathlessness and by pain. The pain is a dull, nagging ache, at times almost intolerable; it occurs in the left submammary region, in the lower sternal region, radiates to the left arm, sometimes only to the medial epicondyle, sometimes down to the fourth and fifth fingers. On one occasion it radiated to the right arm, and it is definitely made worse by exertion. In a recent attack she had slight pain only below the left breast, but was almost beside herself with pain in the whole of the left arm, which in the course of a week had subsided, leaving a bruised sensation in the region of the pisiform bone. There has never been any suggestion of *angor animi*.

As another example we have Mr. T.S. (Case LVIII), showing mammary pain, as well as retrosternal, associated with effort but not with *angor animi*, and ending fatally.

This man, aged seventy-eight years, was seen on December 16, 1931, complaining of pain along the left costal margin, which radiated to the left arm, a stabbing pain near the apex with every heart beat, retrosternal pain and a dull ache deep to the left scapula, as if due to a heavy weight. These symptoms had been present for a year, growing more severe, and were increased by exertion. Physical examination revealed a heaving apex beat four inches out in the sixth space, an accentuated aortic second sound, apical and basal systolic murmurs, some congestion of the bases of both lungs and of the liver, and œdema of the legs. Eleven days later he died and autopsy revealed slight pericardial adhesions at the base, slight atheroma of the aorta and of the coronary arteries, which were, however, quite patent; and "the myocardium appeared in good condition".

A search through contemporary text-books revealed opinions as varied as they were unsatisfactory, and it was quite plain that on this subject there was much loose thinking. From the "cardinal and crucial symptoms of *angina pectoris* in its true austerity" as laid down by Allbutt[1] to the sweeping statement of Mackenzie[2] that all such pains are either stepping stones on the path to full-blown angina or secondary angina, there range many definitions, which would constitute a large series. If we go back to the description by Heberden,[3] we find that he was very definite. As he told the Royal College of Physicians of London, *angina pectoris* was:

a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare. The pulse is, at least sometimes, not disturbed by this pain. I have seen nearly a hundred people under this disorder, of which number three have been women. *Angina pectoris* belongs to the class of spasmodic complaints for first, the access and recess of the fit is sudden; second, there are long periods of perfect health; fourth, it is increased by disturbance of the mind; fifth, it continues many years without injury to the health; seventh, during the fit the pulse is not quickened; and lastly, its attacks are often after the first sleep.

Whatever confusion has arisen since, after considering these features I think it will be agreed that, first, *angina pectoris* as conceived by Heberden was paroxysmal; secondly, it did not include attacks of cardiac infarction; and thirdly, in view of points two and five, it seems unlikely that it includes cases of coronary sclerosis with steadily decreasing myocardial reserve.

If there be a disease *angina pectoris*, a suitable adjective would be anginal; but a very brief review of journals, text-books and systems reveals many allied terms. One finds pseudo-angina, minor angina, vasomotor angina, phantom angina, stenocardia, mock angina, *angina decubitus*, *angor nevrosique*, anginoid, anginous and anginiform pain; and there are others. The origins of some of these are uncertain and probably unimportant. Others have been devised by leaders of our art, as, for example, sternalgia by Baumes, of Montpellier, stenocardia by Brera, vasomotor angina by Nothnagel, and vasovagal attacks by Gowers. It seems plain that, since Heberden introduced the term *angina pectoris* in 1768, there have been thinkers who realized that there were certain types of pain, apparently of cardiac origin, which did not fit Heberden's description. They invented terms which stressed the similarity rather than the difference. Their efforts were directed towards modifying the only existing definition of a type of cardiac pain rather than towards making new ones; and the result was a stretching of Heberden's original conception till its content became chaotic. More than a century later Allbutt was aware of this confusion. Much of his life work was the elaboration and exposition of an hypothesis of *angina pectoris*; with all his elegance of style and majestic marshalling of arguments, he was too sound a logician to omit a solid foundation of definition for the edifice of his thesis. His views on the causation of angina are at present forgotten or made light of; but, whether they be right or wrong, he did great service in pointing out the confusion which existed in its nomenclature. We are apt to neglect this contribution. Twenty years have passed since the publication of his *magnum opus*, but the confusion remains. The more widely one reads at present, the less sure does one become whether *angina pectoris* is a disease, a symptom-complex or a single symptom. It is perhaps wise to quote here Allbutt's description[1] of angina:

1. Peculiar and startling pain, usually sternal in seat, but, as it forces its path into contiguous segments of the spinal centre, spreading thence into widening areas . . . . 2. Arrest or restraint, brief but imperious, of respiration; and more or less of all muscular activity. 3. Sense of impending death, not constant but in many cases appalling; often, even in slight or incipient cases, an organic dread or sense of ill omen, as contrasted with rational apprehension.

And he adds as subsidiary or accidental features high arterial blood pressure, arterial disease, vasomotor disorders, upset of the heart's rhythm and sudden death, and mentions aberrant and larval cases.

The mention of only a few descriptions since his book was published will show how his teaching has been disregarded. In Conybeare's text-book,[4] published in 1929, we meet again the dreadful term pseudo-angina, which is described as pain without

any discoverable lesion; minor angina, which is pain, not very severe but definitely induced by effort; *angina major*, a brief, excruciating paroxysm with ashen grey facies and immobility; and *status anginosus*, which is explained as due to cardiac infarction. Every item of this description would have met with Allbutt's disapproval. In Osler and McCrae's "Modern Medicine"[5] the problem is dismissed with a suggestion that spasm of the coronary arteries is probably a frequent factor. In "Heart Disease", by Paul D. White,[6] we find on different pages *angina pectoris* referred to as "this dangerous symptom", "an end result of hypertension", and "a complication of cardiac enlargement". Crighton Bramwell,[7] in one of the most recent publications in English, defines angina as "a paroxysmal cardiac pain"; yet no less an authority than Dr. John Parkinson,[8] speaking at the British Medical Association Centenary Meeting, says: "*Angina pectoris* is a serious disease of the heart." Perhaps the best single example of this diversity and confusion of current opinion is to be found in an article by Cowan,[9] in which he writes in line 1: "*Angina pectoris* is . . . merely a single symptom of varied origin." And in line 11: "In angina pain is the dominating symptom."

It appeared, then, after a review of the development of the concept of *angina pectoris*, that we knew cardiac infarction clearly; that *angina pectoris*, having been described by Heberden, had been confused for one hundred and fifty years till Allbutt attempted to lay down clear criteria for it, with so little effect, however, that today, twenty years later, opinions still differ as to whether it is a disease or a symptom. Beyond these two—assuming for the moment that there is such a disease as angina—there were many examples of pain, presumably of cardiac origin, as baffling in their similarity as in their differences, which were left out of consideration or lumped together as "the pain of chronic heart disease", heartache, *Herzschmerz*, pseudo-angina, and so on. It is not without significance that the only kind of cardiac pain so far satisfactorily set apart bears a name which makes no reference to angina, but which does make plain the underlying pathological mechanism—cardiac infarction, due to coronary obstruction. Further, in this disease the only characteristic features of the pain are its duration and severity; even these are non-essential, as the catastrophe may be painless,[10] and the diagnosis depends for its confirmation on such other features as auscultation, temperature, blood pressure and electrocardiogram. In short, an attempt to define *angina pectoris* seemed to indicate the necessity for classifying cardiac pain.

A classification may be regarded as an exercise in logic, which requires insistence on definite terms; and a few will be laid down here, at least for the purposes of this paper. There is as yet no theory of *angina pectoris*: "A general proposition is not a theory until its ascendancy is generally granted" (Allbutt[11]), and, as we have seen, none of the hypotheses or views current of the nature or causation of angina has yet reached this standard. An inaccuracy which has caused much vagueness in the records of such cases as we are dealing with, is the use of the term precordia. As Allbutt[12] points out, this originally referred to the epigastric region and a part of the diaphragm; today it is taken to mean generally that region of the chest wall lying in front of the heart. Such a definition may include pain behind the upper, middle or lower part of the sternum, parasternal pain, submammary pain, or even pain in the left axilla. But the areas of orthodox surface anatomy, such as the hypogastrium or femoral triangle, do not change their limits with alteration in the position or size of adjacent organs; so far as possible, then, the term precordial will be avoided. Again, substernal is an adjective which should be used with care. Pain of cardiac origin does occur in the region of the xiphisternum, in the epigastrium and in the neighbourhood of the umbilicus; but, if so, it should be correspondingly recorded. As generally used, substernal pain means pain deep to the sternum; this is casting aside the standard anatomical position and adopting that used by gynæcological patients who complain of "high stomach". How widely the situation of pain may vary is instanced by the following four patients.

Mr. G.M., at the age of sixty-three, with nothing notable in his previous history, suddenly developed pain behind the lower end of the sternum while walking upstairs; he stood still; the pain lasted a few minutes. There was no radiation and no hyperalgesia. In spite of frequent recurrences he drove his cab for a week, but finally he had to give in and went to hospital, where he was first seen by a surgeon and was examined by X rays for gall-bladder disease. He was subsequently diagnosed as suffering from cardiac infarction and died suddenly two weeks after admission to hospital and six weeks after the initial attack.

Mrs. M.M., at the age of fifty-five, gradually developed swelling of the feet, breathlessness on exertion, bad dreams and gasping starts from sleep, and pain under the left breast. Three weeks later she complained for the first time of pain in the left parasternal region, at the level of the third to the fifth costo-chondral junctions.

Mrs. S.M., with a strong family history of heart disease, but no previous illnesses herself, at the age of fifty noticed slight breathlessness. At the age of fifty-one she began to notice that she had to stop three or four times on a hill from pain in the left side of the upper part of the chest, spreading to the neck and inner left arm, and a choking feeling in the suprasternal notch. Subsequently she suffered a severe attack of pain to the left of the sternum, between the scapulæ and down the inner left arm, which subsided (after admission to hospital as suffering from coronary thrombosis) to pain and hyperalgesia in the region of the left scapula.

Mr. C.H., with a negative history but strongly positive Wassermann reaction, at the age of sixty-three began to notice breathlessness accompanied by pain in the region of the umbilicus, always caused by exertion and relieved by rest. His electrocardiogram was strongly suggestive of coronary thrombosis.

It will be noted that six different sites of pain are recorded in four persons. This variation must have some significance, but we shall never learn to appreciate it until the differences are accurately recorded along with their full clinical and *post mortem* context. As Hilton[13] said seventy years ago, "accurate information respecting the exact seat

of pain is a very important step towards obtaining a correct diagnosis". In attempting clarity, even the title of this paper is open to criticism; what, it may be asked, is meant by cardiac pain? One can only answer: pain which, from a consideration of all its features, one decides is directly associated by way of the central nervous system with a disturbance, functional or organic, of the heart. This presents us with the difficulty that the *angina pectoris* of Allbutt would be not cardiac pain, but aortic; this problem will arise in another form elsewhere. Pain, of course, is a symptom, nothing more and nothing less. We have to answer the question: Does cardiac pain of anginal type represent the outstanding symptom of a clearly definable disease with individual pathology? Or: Is it a symptom-complex of various origins and capable of elaboration in varying degree, like the pain of renal colic, which, while preserving the main features of colic and genito-urinary distribution, may vary in site, intensity, extent of radiation, duration and prognosis accordingly as the obstruction is congenital or traumatic, due to stone or clot, high or low, fixed or movable? Let us consider two somewhat similar problems. Banti's disease (as it has come to be called, though Banti confined himself to calling it a symptom-complex) is still a subject for argument. Cases due to malaria, syphilis and other known disorders have been separated out; a number of cases remain which fit Banti's description and the pathogeny of which is unknown. Will further knowledge distribute these too under existing headings or will they be grouped under a new one? Again, and more nearly parallel, consider epilepsy. The falling sickness has been known since the earliest historical times. At present we recognize the epileptiform fits of lead poisoning, cerebral crises of arteriosclerosis, cerebral tumour and so on; but there is still a group which we term idiopathic epilepsy, in which no specific lesion, beyond degenerative changes which are considered to be of a secondary nature, is found *post mortem*. Is there a metabolic dyscrasia which causes fits and which will ultimately be termed epilepsy, while similar convulsions in other disorders are not unjustly described as epileptiform? The question obviously arises: Is there a condition, *angina pectoris*, of unknown causation and with no characteristic pathological picture, compared with which the pain in cardiac infarction or failing coronary circulation or neuro-circulatory asthenia will fairly be described as anginiform?

The philosophical significance of pain does not greatly concern us here; nevertheless we may recall Hilton's[14] words: "Under injury, pain suggested the necessity of, and indeed, compelled him to seek for, rest." This is lost sight of in those modern operative procedures which relieve anginal pain, while leaving the cause untouched. Who would dream of a similar procedure in renal colic? This reminds us that in some cases pain, instead of imposing quiescence on the sufferer, stimulates him to restless and even violent movement. Are these different reactions purposively related to the type of lesion in question (restless movements may well dislodge an impacted calculus, raise blood pressure and open up accessory circulatory paths, while immobility will minimize friction between raw surfaces and lower blood pressure); or are they merely dependent on severity and personal idiosyncrasy? In 1863 Hilton[15] wrote:

> Pain in any part, when not associated with increase of temperature, must be looked upon as caused by an exalted sensitiveness of the nerves of the part, and as a pain depending upon a cause situated remotely from the part where it is felt. I would ask you to regard such pains as resulting from some direct nervous communication passing between the part where the pains are expressed and the real and remotely situated cause of the pain.

Beyond this general principle the work, excellent as it is, of Ross,[16] Head,[16] Mackenzie[17] and Morley[16] has not carried us very far. The irreducible basis of the cognition of pain is, of course, a stimulus, a sense organ, a lower level path in the central nervous system and a connexion with the higher centres. At the very outset we are faced with the question: what is the adequate stimulus? The skin has its specific receptors for pain, heat, cold, touch, and so on; and it is agreed that the stimulus in hollow abdominal viscera is none of these, but tension. Perhaps the view most widely held is that the stimulus is ischæmia of the myocardium. Sir Thomas Lewis,[18] in his St. Cyres Lectures for 1932, has recently suggested another explanation; and there is yet a third: no one who has worked with local anæsthesia can have failed to remark that, when all other tissues have been rendered insensitive, nipping an artery with forceps can still produce pain; so that arterial walls are apparently very sensitive. We have seen, and shall see in more detail, that pain, induced by effort and of the same type, may occur at different times and in different positions in the one patient. Are we then to assume that the lesion varies correspondingly from time to time? If so, what kind of lesion can it be? One more difference, and a very striking one, remains to be noted; it is known that amyl nitrite gives relief in certain conditions which are termed *angina pectoris*, and that it does not relieve the pain of cardiac infarction. But it should be further noted that there are certain cases of pain, definitely due neither to cardiac infarction nor to classical *angina pectoris*, severe, prolonged and associated with organic heart disease, which amyl nitrite does not relieve—for example, Mr. P.D. (Case VIII). In what way do these cases differ in causation while otherwise remaining so similar?

In view of the foregoing, it was decided to collect one hundred consecutive cases of cardiac pain and to analyse them as fully as possible in view of the above problems. These hundred cases which will now be dealt with come from several sources. The majority are from Sydney Hospital, more out-patients than in-patients, and my thanks are due to all those physicians on the staff who have allowed me access to these. A few at the Royal Prince Alfred Hospital have come to my notice

through the kindness of Professor Lambie; a few are private patients of my friends; and the remainder are from my own out-patient department at the Royal South Sydney Hospital. They are not consecutive in that I omitted no case of cardiac pain in series from any of these sources; sometimes I was otherwise engaged and in eighteen months missed quite a number. But as they came to my notice they are consecutive and quite unselected. The only way in which they fail to constitute a random sample is that the great majority are public hospital patients and very few come from private practice.

Plan of Analysis of Cases.

The plan of analysis of the cases is as follows:

1. Age when first seen, set out in Table I.
2. Sex.
3. Occurrence of pain as first symptom.
4. Age at the onset of pain (Table II).
5. Incidence of relevant diseases in the immediate family history (Table III).
6. The characteristics of the pain:
   - A. Duration—
     - (i) Frequency of durations of pain for whole series.
     - (ii) Frequency of durations in patients with one type only (Table IV).
     - (iii) Association of causal disease and duration of pain.
   - B. Site (Table V).
   - C. Associated features—
     - (i) *Angor animi* (Table VI).
     - (ii) Sense of constriction.
     - (iii) Hyperalgesia.
     - (iv) Cramps in lower extremities.
     - (v) Dyspnœa.
     - (vi) Electrocardiographic changes (Table VII).
     - (vii) Presence of definite cardiac disease.
     - (viii) Factors aggravating and/or inducing pain:
       - (*a*) Emotion.
       - (*b*) Exertion.
       - (*c*) Cold.
   - D. Subjective aspects of pain (Table VIII).
7. Presence of other diseases—
   - A. Gall-bladder disease.
   - B. Spondylitis (Table IX).
   - C. Miscellaneous (Table X).
8. Ætiological groups—
   - A. Hypertensive heart disease (Table XI).
   - B. Chronic rheumatic carditis (Table XII).
   - C. Cardio-aortic syphilis (Tables XIII and XIV).
   - D. Chronic nephritis (Table XV).
   - E. Cardiac infarction (Tables XVI and XVII).
   - F. Myocardial degeneration (Table XVIII).

   Summation of ætiological groups according to sex (Table XIX).
9. Deaths (Table XX).
10. Average duration of life since onset of pain, in ætiological groups (Table XXI).
11. Other aspects—
    - A. Response to pain—motionless or restless (Table XXII).
    - B. Cases not showing cardiac enlargement (Table XXIII).

Analysis of Series of One Hundred Unselected Patients Complaining of Pain in Association with Heart Disease.

Age When First Seen.

The age when the patient was first seen is shown in Table I. The patients are distributed in decades according to sex. It will be noted that the maximum incidence of female patients is grouped about the period of the menopause, while the excess of male patients is made up in the later years of life. This table represents the patients as they were first seen in the course of investigation.

Table I.

Age Period in Years.	Males.	Females.	Total.
11 to 20	2	—	2
21 to 30	2	—	2
31 to 40	6	7	13
41 to 50	10	11	21
51 to 60	15	23	38
61 to 70	16	1	17
71 to 80	7	—	7
Total	58	42	100

Sex.

Of the 100 patients, 58 were males and 42 were females.

Occurrence of Pain as First Symptom.

Pain appears to have been the first symptom in 40%, to have developed coincidently with breathlessness in 19%, and to have been a subsequent development in 41% of patients.

Age at Onset of Pain.

The age at the onset of pain, which is shown in Table II, does not coincide with the age when the patients were first seen; but again the females are grouped in the menopausal period.

Table II.

Age Period in Years.	Males.	Females.	Total.
1 to 10	1	—	1
11 to 20	2	—	2
21 to 30	2	1	3
31 to 40	6	9	15
41 to 50	12	15	27
51 to 60	19	16	35
61 to 70	9	1	10
71 to 80	6	0	6
81 to 90	1	0	1
Total	58	42	100

Relevant Disease in the Family History.

The incidence of relevant disease in the immediate family history is shown in Table III. In this table a cross represents one member of the family (parent, uncle, aunt, brother or sister) who suffers, or has died, from cardio-vascular disease, nephritis or gout. It will be seen that no less than forty-six, or nearly half, give a positive family history; and the average is far nearer two than one affected relatives. Moreover, this is undoubtedly below the real total, because from such patients the most incomplete family history, or even none, is frequently available. Twenty-one were males and twenty-five females. In rheumatic cardiac disease Paul D. White[19] gives the figure as from 33% to 50%, but I found no definite statistics for other disease groups.

TABLE III.

Case Number.	Sex.	Affected Relative.	Case Number.	Sex.	Affected Relative.
VI	M.	× ×	LXVII	M.	×
VII	M.	×	LXX	M.	×
X	F.	× × ×	LXXI	M.	×
XII	F.	× ×	LXXII	M.	× ×
XIII	M.	×	LXXIII	F.	×
XIX	F.	× × ×	LXXIV	F.	×
XX	F.	× × ×	LXXV	F.	× ×
XXI	F.	×	LXXVII	M.	×
XXVI	F.	×	LXXIX	M.	×
XXIX	M.	× × × ×	LXXX	F.	× × ×
XXX	M.	×	LXXXI	F.	×
XXXII	F.	× × × ×	LXXXII	F.	× ×
XXXIV	F.	× ×	LXXXIII	F.	×
XXXV	M.	×	LXXXIV	M.	×
XXXVI	F.	×	LXXXVII	F.	× ×
XXXVII	M.	× ×	LXXXVIII	F.	×
XXXVIII	F.	× × ×	XC	M.	×
XXXIX	F.	× × ×	XCI	F.	× × ×
XLIX	M.	× ×	XCII	M.	×
LIV	F.	× ×	XCIII	F.	×
LV	M.	× ×	XCIV	M.	×
LXV	M.	×	XCVIII	F.	×
LXVI	M.	× ×	C	F.	×

The Characteristics of the Pain Experienced.

Duration.

Sometimes the pain lasted for over twenty-four hours, when it was noted as of days' duration; sometimes it lasted for periods ranging from fifteen minutes up to twenty-four hours, when it was put down as lasting for hours; and there were briefer attacks ranging from a momentary stab to one or two minutes, up to a quarter of an hour—these were recorded as lasting for minutes. A certain proportion of patients suffered pain of one kind of duration only—more than half the series; but the remainder had pain at one time of one length, at another time of another length, the different kinds of pain occasionally being superimposed one on another. The distribution of the types of pain is shown in Table IV. From this table it will be seen that in a small but definite majority of instances the pain exceeds the limits of definition of a brief paroxysm. It should be noted that the briefest pain can be agonizing and of extensive radiation; that "pain that lasts can be borne" and may remain localized; while both may be associated with definite and severe organic disease, for example, Cases XLVIII and LXVII respectively. In Case XLVIII, with advanced mitral regurgitation (rheumatic),

TABLE IV.

(i) *Frequency of durations of pain for whole series:*

Minutes (up to 15 minutes)	68	
Hours (up to 24 hours)	59	83
Days (over 24 hours)	24	

(ii) *Frequency in patients with one duration only:*

Minutes	31
Hours	20
Days	6

(iii) *Association of disease and duration of pain:*

Of the 31 patients suffering pain of minutes' duration only,
- 12 had chronic rheumatic carditis
- 8 had hypertensive heart disease
- 6 had syphilitic lesions
- 3 were classified as myocardial degeneration
- 1 suffered from chronic nephritis with secondary hypertension
- 1 had cardiac infarction
- 5 showed hypertension as a secondary feature.

Of the 20 patients suffering pain of hours' duration only,
- 11 were primarily hypertensive
- 3 had chronic rheumatic carditis
- 3 had syphilitic lesions
- 2 were classified as myocardial degeneration
- 1 had chronic nephritis
- 4 had hypertension as a secondary feature.

Of the 6 patients suffering pain of days' duration only, all were cases of hypertension.

mid-left para-sternal pain spreads to the left side of the neck, left breast and left costal margin, but disappears in three minutes when the patient rests, while in Case LXVII, in which the patient has had cardiac infarction, the pain is localized to the mid-sternal region and lasts for three days. In the third section of the table it is seen that the longer the duration of pain, the more frequently is hypertension the predominant feature of the case; though it is by no means uncommon in pain of briefer duration as well.

Site.

We pass now to what should be one of the most striking features in surveying the series, namely, that, in recording carefully the site of pain on serial occasions over a period of months or years, we find individual patients experiencing pain in divers places, while retaining such general features as aggravation by effort, association with palpitation and breathlessness and hyperalgesia. Following on the introductory remarks about the inadequacy of the term precordia, the pain in this series of patients has been carefully observed, and Table V shows the various sites in which pain has been recorded, and with what frequency. The only other author in whom I have found a consistent attention to similar detail is K. Shirley Smith.[20] These sites, of course, were in many cases connected by radiation, as in Case LXXVIII.

On October 18, 1933, the patient complained of attacks of pain starting behind the mid-sternum and spreading out to the axilla and left side of the neck, lasting from a few minutes to an hour, five or six times a day; the pain came on both with exertion and at rest.

It also happened, however, that pain on occasion was present in outlying sites alone, as is shown in the following extracts from case histories.

Case X. March 9, 1932: The patient feels much easier; pain is now localized over the left pisiform bone.

Case LXVIII. October 27, 1930: The patient has complained of pain in the left fingers for some days. November 1, 1930: The patient now has pain in both arms.

Case LXI. January 18, 1933: The patient complains of pain along the right border of the sternum, gradually increasing for two weeks; two days ago a sudden severe exacerbation occurred; this was accompanied by dyspnœa.

It will be noted, in this series at any rate, that simple mammary pain was a rare finding (the rarest of left-sided occurrences); that when it did occur it was associated with definite organic disease; and that, taken over the whole series, the mammary site was recorded in only 19% of all instances. Attention should also be drawn to the comparative infrequency of radiation to the fingers, and also to the surprising frequency of pain along the left costal margin—in more than 25% of the cases. Nevertheless, there seems to be good reason for associating this site with cardiac lesions, as for example in the following cases.

Case XXXIII. December 30, 1931: While sitting in a picture show the patient was suddenly seized with pain below the left costal margin; it was like a red-hot iron (cardiac infarction).

Case XLVI. In this case the pain came on suddenly, was very severe in the epigastrium, under the left breast and along the left costal margin.

The first of these cases shows costal margin pain as a solitary manifestation; the second shows it in association with other, commoner sites. These different and varying sites will be discussed again later; it suffices to reiterate here that they are presumably not all referred from a lesion in the one position.

TABLE V.

Incidence of Pain in Different Sites for the Whole Series, at all Times.

(i) Retrosternal and immediately left parasternal:			
Upper (costal cartilages 2 and 3)	10		
Middle (costal cartilages 4 and 5)	31		
Lower (costal cartilages 6 and 7)	56		
		—	97
(Retrosternal only: 9.)			
(ii) Left mammary			63
(Mammary only: 4, all had definite organic disease, and two died suddenly.)			
(iii) Outlying sites:			
Left-sided:			
Left arm	43		
Left costal margin	27		
Left fingers	16		
Left shoulder	14		
Left side of neck	12		
Left axilla	10		
Left side of head	6		
		—	128
Mid-line:			
Epigastrium	13		
Interscapular	8		
		—	21
Right-sided:			
Right arm	11		
Right shoulder	9		
Right parasternal	2		
Right side of head	1		
Right fingers	1		
		—	24
Total		—	173

Features Associated with the Pain.

Angor Animi.—In no case was the *angor animi* an inevitable feature of the attacks of pain in a given subject; in only one case did it accompany a brief paroxysm. It should be noted that throughout the investigation care was taken never to suggest angor by a leading question. In all the cases shown in Table VI the information was given voluntarily in answer to a request for a general description of the pain.

TABLE VI.

Case Number.	Sex.	Disease.	Duration of Pain.	Present State.
VII	M.	Old cardiac infarction; ? previous hypertension.	Minutes.	Alive.
XXVIII	F.	Advanced hypertensive heart disease.	Hours.	Alive.
XXXI	F.	Hypertension.	Days.	Alive.
LIII	F.	Cardiac infarction, hypertension and positive Wassermann.	Days.	Alive.
LXVIII	F.	Cardiac infarction, hypertension.	Hours.	Dead.

Sensation of Constriction.—A sensation of constriction or of a heavy load on the chest was present in a further 21% of the cases.

Hyperalgesia.—Hyperalgesia, either accompanying or following the pain, developed sooner or later in thirty-six cases, or 37%; of the patients, fifteen were men and twenty-one women—42% and 58% respectively; from sixty-one, or 63%, it was absent; in the remaining three cases it was not recorded. When it was present, it was a very definite phenomenon, as in the following cases.

Case XXXV. February 7, 1932. Sometimes when the patient has the pain the back of his shoulders feels sore, as if his clothes were dragging.

Case XLV. March 30, 1932. He sometimes has sufficient hyperalgesia to make a tight singlet uncomfortable.

Case LII. January 18, 1933. The patient complains of a tender spot over the seventh rib in the left anterior axillary line; he has had no retrosternal pain recently; he says that breathlessness pulls him up before the pain comes on.

Cramps in the Lower Extremities.—Cramps in the lower extremities were present in thirty patients, absent from fifty-six and not recorded in fourteen. Of the 35% with cramps, 73% also had hypertension, and 60% were men.

Dyspnœa.—Dyspnœa in varying but definite degree was present in all but four of the hundred cases, and, as shown earlier, in 41% it was the first symptom to appear. Let us consider the four cases in which there was no complaint of dyspnœa.

In Case XCVII electrocardiographic changes were present and the patient's pain was accompanied by a sense of constriction.

In Case XCIV the patient has hypertension and Buerger's disease, which pulls him up after he has walked a hundred yards; so far he has had only the one attack of cardiac pain.

In Case XLI the diagnosis was presumably toxic myocardial degeneration. The patient had pain, hypertension, spondylitis and cholecystitis, and was relieved by the treatment of severe dental sepsis.

In Case XVII the patient died of definite cardiac infarction two months after the onset of pain; in the six weeks before his admission to hospital he continued his activities as a cab-driver, limitation being imposed by pain before breathlessness developed.

There are other cases of this, for example Case LX; and the reverse, dyspnœa bringing activity to a stop before the development of pain, may be seen in Case LII and others.

Electrocardiographic Changes.—Electrocardiographic abnormalities were recorded in sixty-

seven out of eighty cases, or 83%. These changes included left axis deviation, right axis deviation, all grades of heart block; auricular fibrillation; classical Pardee curves and various alterations in the *S-T* interval or *T* waves, interpreted as evidence of myocardial degeneration. In general, however, the electrocardiographic changes were characteristic of the respective disease conditions observed clinically. There was nothing which could be specifically associated with a liability to experience pain. In thirteen cases the electrocardiogram showed no departure from normal; these are shown in Table VII.

TABLE VII.

Case Number.	Clinical Findings.
XVI	Definite cardiac infarction clinically—fall in pressure and friction rub.
XX	Chronic rheumatic carditis; aortic regurgitation; hypertension; lower retrosternal pain.
XXI	Hypertensive heart disease.
XXXIII	Hypertension. Clinically cardiac infarction.
XXXIX	Chronic rheumatic carditis; mitral regurgitation; hypertension; Addisonian anæmia. Pain: retrosternal, mammary, left arm and fingers, of minutes' duration.
XLI	Toxic myocardial degeneration. Hypertension, cholecystitis and spondylitis.
XLVI	Hypertensive heart disease.
LIII	Syphilis; hypertension; clinical cardiac infarction.
LXXIII	Gross hypertension; old cerebral thrombosis.
LXXV	Syphilis; hypertension.
XCI	Syphilis; chronic rheumatic carditis; mitral regurgitation.
XCIV	Hypertension. Buerger's disease.
XCV	Chronic rheumatic carditis; mitral regurgitation.

Presence of Definite Disease.—Definite evidence of cardiac and/or aortic disease was present in every case.

Factors Aggravating and/or Inducing Pain.—Here are considered the aggravating effects on pain of certain factors: (*a*) Exertion produced or aggravated the pain in all but three cases, that is, 97%. (*b*) Emotion produced or aggravated it in thirty-two, or 34%, and failed to do so in sixty-three, or 66%. Looking at these figures in another way, we see that emotion is admitted as having an adverse effect in 22·5% of the men and in 45% of the women. (*c*) Cold caused or increased pain in only 5%.

Subjective Aspects of Pain.

Having dealt with these more objective aspects of cardiac pain and its associated features, it is perhaps only right to glance at its more subjective side. The patients' own descriptions of pain are many and varied. Some of these are given in Table VIII, as nearly as is reasonably possible in the patients' own terms. Though the table is not complete, that is to say, it does not include every case nor each type of pain in each patient mentioned, it will be observed that it has been set out in some detail. No apology should be needed, for the pain is what troubles the patient, and of it we should have a sympathetic understanding. It is a fruitful source of errors in diagnosis, and an examination of Table VIII, together with Table V, should be sufficient reason for avoiding the vague and inaccurate term precordial. The table shows that pains were variously described—like a pinprick, stinging, stabbing, weird and sharp, tearing, gnawing, burning, dull, dragging, vice-like, accompanied by an indescribable feeling of dread, slight, and nagging. Site and duration have already been dealt with in Tables IV and V. It should be noted that sometimes the onset is acute and sometimes ingravescent; and similarly with the termination. Above all that the pain, for the most part, is imperious; for example, in the following cases:

Case XXVIII. The pain was momentary; but the patient had to sit down.

Case XXXV. The patient felt that if he had gone on, it would have got worse; but he couldn't have gone on.

Case XLV. The patient, who is battling on with light wharf labouring, says that when he does get pain he has to stop till it eases.

Case XXXIII. The patient left his tram and, reaching a doorstep, just sat till someone moved him to hospital.

The pain may make boys look agonized, though they try to hide it, and strong young men weep, set elderly women beside themselves, and cause grown men to shriek aloud. Yet it can be far less severe, while retaining its imperious nature, and its severity is no safe guide to the gravity of the lesion responsible.

TABLE VIII.

Case Number.	Subjective Description of Pain.
I	It is not agonizing, but she has to stand still; accompanied by a choking sensation and followed by palpitation.
III	Pain on exertion . . . stops when he stops.
V	Pain on exertion . . . when he uses a hammer in the right hand, develops pain in the left arm, which clears up in two to three minutes' rest.
VI	Pain, chiefly on going uphill; when he stopped, the pain stopped. Then developed the duodenal ulcer, which caused quite a different pain.
VII	The pain only lasted a few minutes, but was awful—he nearly went off.
VIII	Gripping pain across the mid-sternum; original pain (mammary) bad, too.
IX	Later when breathlessness arose, anything causing it caused pain as well.
X	Has been beside herself with pain, whole left arm from shoulder to elbow; a week later pain localized over pisiform bone.
XI	Pain is brought on by excitement; has given up attending pictures. Sometimes wakes from dreaming with severe pain.
XIII	It used to hit and squeeze and go away.
XIV	Pain comes on chiefly if he lies on his left side. It is sharp, and lasts about fifteen minutes.
XVI	A dull ache, as if someone were holding him tightly; he wondered what had struck him, he had known nothing like it before; had not known what pain was. He rubbed it, tried to lie down, but had to keep walking about, and cried with the pain.
XIX	The pain was so severe that she vomited. She used to take off her coat because it felt as if it was dragging. After spells of pain the skin was too sore to touch.
XXII	If it comes on exertion he has to stop, and it lasts for hours, or even all day. He also suffers from acute momentary stabs of pain.
XXIII	It was a gripping sensation (lower retrosternal) and he had to stop; at the same time he had cramps in the calves; now the pain attacks him at any time, even without exertion.
XXIV	Noticed for the first time that carrying a tucker bag was too heavy for his left hand. Almost at same time developed retrosternal pain. At first on exertion only. When it's gone, it's gone. Finally couldn't walk across street—just wanted to grasp his chest and stand still. (Later had definite cardiac infarction.)
XXVI	The pain now follows on a sinking feeling during which she feels the circulation in the left arm has stopped. Then she gets relief, and as it starts again the pain comes on.
XXVII	While walking is suddenly gripped by pain over the left nipple—has to sit down and hold his breath. Mother says he appears to be in agony while it lasts. (Boy of thirteen years with rheumatic mitral disease.)

TABLE VIII.—*Continued.*

Case Number.	Subjective Description of Pain.
XXVIII	The pain was sharp and stinging, especially after hard work. It was momentary, but she had to sit down and hold herself. Two years later gripping pain brought on by exertion, and gives her an indescribable feeling of dread. Has to sit still. Two years later an almost constant ache—not severe.
XXX	He has to sit and rest completely, and it takes one to two hours to fade away. He also complains of pain, not at the same time, behind the medial half of the left clavicle and down the left arm as far as the medial epicondyle, heavy and dragging in quality, so that he has to support his left arm.
XXXI	When this pain comes on she has a sense of constriction about her chest, and thinks she may die. Later, severe submammary pain—has to press on her breast while walking.
XXXII	Has a tight, painful, smothering feeling in the left third and fourth interspaces, just adjacent to the sternum, coming on suddenly, when "you've got to keep still". "A weird, sharp pain as if the blood had stopped." She lives up a hill, used to walk half-way; nearly died; then walked the other half. Now has to be taken up in a cart.
XXXIII	The pain was severe, like a red-hot iron; so bad that he left the tram and sat on a doorstep, whence he was taken to hospital. The next morning the pain had changed to a dull ache, and all the clothes and his bedding felt too heavy.
XXXIV	An indescribable pain. She couldn't get her breath, but she had to walk about. It then passed off rapidly, leaving numbness and tingling in the volar aspect of the left forearm and all fingers of the left hand.
XXXV	The pain seemed to catch him very quick; felt that if he had gone on it would have got worse; but he couldn't have gone on.
XXXVII	Momentary pain on walking fast; now goes slowly so that it does not have a chance to develop.
XXXVIII	Rubs her chest for relief. Constant dull submammary pain with occasional sharp stabs, worse on exertion.
XXXIX	"I would get sudden and sharp pin-prick pain, when feeling very tired, lasting only a couple of minutes; at times a dull ache followed for a short time, after which there would be a sense of tenderness and soreness." Extract from a letter.
XL	While walking down the street was seized with pain alongside the upper sternum like a "bolt from the blue". Tried to go on, but had to sit down. Later, it is partly relieved by rubbing.
XLIII	"A burning pain."
XLIV	Could not walk ten feet without feeling as if a dagger was stuck in his chest. The pain was terrific, and made him "holler out".
XLV	Is doing a little wharf-labouring; the foreman lets him go easy; gets pain towards the left lower sternal region; it makes him feel faint, but clears up in a few minutes with rest.
XLVI	The pain comes on suddenly, is very severe, and ceases suddenly. After it passes there is a general soreness of the affected part.
XLVIII	Pain is gnawing in character; grips him and, if he does not stop, radiates widely.
XLIX	The pain comes on as he gets breathless.
LII	The pain is like a "heavy bar".
LIII	"A gnawing pain."
LVI	Pain is like a cramp—comes and goes all night.
LVII	An occasional dull ache—lower sternal; later, dreadful throbbing pains in nipple and behind lower sternum.
LVIII	A dull ache behind the lower sternum, and a stabbing pain near the apex with every beat.
LX	Pain followed by hyperalgesia; could not bear pressure of elastic of bloomers.
LXII	Pain comes on gradually during exertion.
LXV	A fearful attack of pain behind the sternum, and down the arms, where it was even worse.
LXVI	A tearing pain, and he feels smothered; afterwards the skin of his chest feels raw, as if rubbed with a brick.
LXVII	The pain, when it comes, is just "out of the blue".
LXXII	An ache behind the upper sternum and in the volar aspect of each wrist for the last year, on exertion and emotion—if he gets in a "wax" is bad for a whole day afterwards.
LXXIV	A slight mammary ache, dull—lasts for hours.
LXXV	Pain is accompanied by hyperalgesia; can't bear the elastic of her bloomers. She can't get excited, or she nearly dies.
LXXVIII	Stabbing pain under the left breast; rolled about in agony. Lasted for three days till relieved by morphine (cardiac infarction). Subsequently attacks of pain, in which his left chest feels as if in a vice, followed by hyperalgesia; has to take clothes off.
LXXIX	Hyperalgesia relieved by pressure.
LXXXI	Hyperalgesia; can't bear clothes.
LXXXII	First attack of pain awakened her, and she dared not move for three hours.
LXXXVII	Pain comes like a cramp; she can't breathe, and has to hold her left breast; shoots down to each elbow; only momentary, but leaves her exhausted.
LXXXVIII	Pain accompanied by hyperalgesia; cannot bear corsets.
XCII	A gnawing pain.
XCIV	The pain was right inside.
XCVIII	A nagging pain like a toothache; brought on by exertion—then may last all day.
XCIX	Needle-like stabs in region of apex beat.
C	Hyperalgesia; can't stand beads round neck; massages the left arm for relief.

Presence of Other Diseases.

Gall-Bladder Disease.

Definite evidence of gall-bladder disease, obtained either radiographically or by laparotomy, was found in twenty-seven of the patients investigated under this heading; it was absent from thirteen. It is not suggested that a corresponding incidence of 67% would have been found could the whole series have been submitted to examination; but it is suggested that, even so, an occurrence of 27%, or more than a quarter, even as a maximum, is a striking feature and one which might be unexpected in diseases apparently so unrelated. It was, for example, much more marked than the history of appendiceal disease, admittedly a far commoner complaint, but one which was noted only in thirteen cases (including several in which the appendix appears to have been removed as a routine during gynæcological procedures). It must be admitted also that the appendix was not searched for as the gall-bladder was; but it is perhaps not too frivolous to suggest that in these days any suspicion of its disaffection is less likely to have been overlooked than in the case of the gall-bladder.

The presence of gall-bladder disease has been noted by many observers, who express opinions ranging from that of Paul D. White,[21] who merely refers to it in passing as one source of focal sepsis, to T. F. Cotton's,[22] who, in dealing with outside influences on the heart, speaks of the toxic state from an infected gall-bladder, and who gives it pride of place, and to Crighton Bramwell's,[23] who says:

> There is a considerable volume of evidence that gall-bladder infections may give rise to toxic myocarditis, which manifests itself by anginal symptoms. In some cases these symptoms are permanently relieved by cholecystectomy.

On the pathological side it has been shown by C. H. Miller[24] that the incidence of severe atheroma is higher in gall-bladder patients; and Meakins and Eakin[25] found gall-stones the commonest non-vascular association in sixty-two cases of coronary thrombosis. So it is that from the clinical aspect we find R. Dawson[26] referring to the frequency of gall-bladder history in cases of hypertension; and L. E. Hurley[27] urging the necessity for dealing with the gall-bladder before assessing the degree of permanent cardiac damage. Mackenzie[28] held, what is generally accepted, that distension of the stomach or colon with gas may lead to cardiac embarrassment; and it has been shown experimentally by Burgess[29] that tachycardia follows immediately on distension of the stomach, often associated with inversion of the *T* wave and accentuation of the *Q* wave, which he suggests may be due to alteration of the cardiac axis. Roemheld[30] goes so far as to refer to the "gastro-cardiac" syndrome. He attributes this to both mechanical and toxic factors, and states that patients affected by it complain frequently of a dull, painful pressure at the left costal margin, with pain radiating into the left arm. (Costal margin pain was noted in eight of the twenty-seven patients with

gall-bladder disease in this series.) Scott and Ivy,[31] among many other American workers, carried out an experimental study on viscero-cardiac reflexes in frogs and dogs, and showed that bile spillage in the upper part of the peritoneum had a quantitative reflex effect on the heart. As experiments on frogs and dogs give different results, it cannot be said definitely what occurs in man; but one has to agree with the possibility mentioned by them, that a direct viscero-cardiac mechanism may exist; that pathological conditions of the gall-bladder do not occur in all cases of cardiac disease does not exclude it. According to Newman:[32]

> Excessive vagal activity, which may be due to many causes, produces spasm of both gall-bladder and ampulla, and, as the ampulla is by far the strongest muscle in the system, the result is complete stoppage of the bile flow.

This may well be a secondary result of the effect of hypertension on the depressor nerve, which reacts to an increase of pressure by an increase of inhibition; so that one can see the possible establishment of a vicious train of events—hypertension, biliary stasis, biliary infection, toxic effect on the myocardium. Of the gall-bladder patients in this series, 66% had hypertension. So far as relief of pain is concerned, it should be remembered that Weiss and Davis [33] showed that visceral pain could be relieved by infiltrating with local anæsthetics the skin of the segments to which pain was referred, so that removal of a diseased gall-bladder was not an unreasonable procedure. D. Waterston[34] more recently produced a similar effect by the injection of normal saline solution; he wonders whether the effect is due to the removal of Lewis's "*p*" substance; but it is well known that infiltration with saline solution alone will produce an anæsthetic effect.[35]

It is my impression from such patients as I have seen repeatedly, for example, the patient in Case LXXXVII, who suffers from severe flatulence but who was considered too bad an operative risk by both her physician and surgeon, that gall-bladder dyspepsia adds considerably to their cardiac discomfort. In those from whom I have had the gall-bladder removed (Cases LXI, LXXVIII and XCVIII), there has been some amelioration or stay in the progress of their cardiac lesions. At the same time it must be admitted that there are sundry others, for example the patients in Cases XV and LX, whose gall-bladders were removed before they came under my observation, in whom the cardiac lesion is progressing at present, however it may have been temporarily delayed previously.

Spondylitis.

It has been found possible to examine with X rays forty-two patients for spondylitis; of these, thirty-three, or 78·5%, showed lesions. Their ages ranged from thirty-five to seventy-seven years and averaged fifty-five; the average for males, of whom there were eighteen (31%), was fifty-nine years; and the average for females, of whom there were fifteen (36%), was fifty-one years; these may be compared with the average ages for the series of fifty-four and a half and fifty respectively. While, owing to reasons of finance, health and hospital routine, it was not possible to examine both gall-bladder and spine in every case, in twenty-three cases this was done; and both were positive in fifteen, or 65%. There is another point to be remarked in regard to spondylitis, and that is its distribution which is briefly set out in Table IX. It will be seen that of the thirty-two patients whose X ray reports are noted, twenty-two, or 67%, show involvement of the cervical area, and in fifteen of these pain was experienced in the arm and/or hand. In twenty-four out of twenty-nine cases in which the corresponding dorsal vertebræ are affected there is recorded pain along the left costal margin, in the submammary region or in the axilla. Also in Cases X, XXVI and LXXXVIII areas of particular interest from the point of view of distribution of cardiac pain are picked out in isolated fashion. A positive association in more than 75% of cases would appear to indicate more than a casual connexion between the site and occurrence of spondylitis and the localization of pain. The average duration of life since the onset of pain in the spondylitic cases is barely over three years—a full year less than that for the whole series. Of the cases in which spondylitis was recorded, fourteen showed hyperalgesia and seventeen patients denied it, which is a higher percentage (positive) than in the series as a whole. (I have noted no references concerning the association with heart disease of spinal lesions, except in cases of gross curvature.)

TABLE IX.

Case Number.	Site of Spondylitis.	Site of Pain.
I	Thoracic.	Submammary.
II	Lower cervical, thoracic and lumbar.	Costal margin.
V	Cervical and thoracic.	Left arm and axilla.
VIII	Cervical and dorsal.	Costal margin.
X	Between C. 7 and D. 1.	Left arm and fingers.
XIII	Thoracic and lumbar and costo-vertebral joints.	——
XIV	Cervical and thoracic.	Mammary and axillary.
XVIII	Cervical and thoracic.	Mammary.
XXI	Thoracic.	Girdle pain and left arm.
XXII	Cervical and thoracic and upper lumbar.	Costal margin.
XXV	Thoracic.	Costal margin and left hand.
XXVI	C. 6 and 7 and D. 6 and 7.	Left arm and submammary.
XXIX	Lower cervical and thoracic.	Left arm and hand, mammary.
XXX	Cervical and mid-thoracic.	Left arm, costal margin.
XXXVI	Lower dorsal, upper lumbar.	Costal margin.
XLI	Early thoracic.	Lower left parasternal.
XLV	Upper dorsal.	Costal margin.
L	Lower dorsal.	Submammary.
LII	Cervical and dorsal.	Both arms and costal margin.
LV	Cervical and upper thoracic.	Left arm.
LVI	Cervical and lower thoracic.	Costal margin.
LX	Lower cervical and upper dorsal.	Left arm.
LXXII	Dorsal.	Costal margins.
LXXIII	Thoracic and left cervical rib.	——
LXXVII	Lower cervical and thoracic.	Costal margin, both palms.
LXXIX	Cervical and thoracic.	——
LXXX	Lower cervical and upper dorsal.	Submammary, both arms.
LXXXI	Cervical and thoracic.	Axillary.
LXXXVIII	C. 6 and 7.	——
XLVI	Lower cervical.	Left arm.
XCVII	Cervical and thoracic.	Left arm, axilla.
XCIX	Lower cervical and thoracic.	Submammary.

Miscellaneous.

It will be seen that in these thirty patients, who, while under observation, manifested other ailments, there is a thread of relationship. An exception may be made in the accident of concussion in Case XCVII, and perhaps not even there, for the patient was a very old man and it was during his enforced rest that cardiac pain increased to a degree sufficient to make him seek advice. The effect of chronic nephritis in four cases, in promoting increased blood pressure and cardiac hypertrophy, is obvious. The two cases of *diabetes mellitus* are of interest, partly because of their relationship as common results of an arterial occlusion cause, partly because of their potential effect as an aggravating factor in pain. Several references have been made to the latter association in recent journals. It should be mentioned that in both these cases the disease was discovered in my routine investigation while the patients were under treatment for the cardiac condition. One of them (Case LXXXV) had evidence of a cardiac infarction; the other (Case IV) had for long suffered from hypertension. It will be noted that the incidence of *diabetes mellitus* among the patients with cardiac infarction in this series is far less than that recorded in America by Levine.[36] In view of the recent opinion that diabetes should be regarded as a cause of arteriosclerosis, as it undoubtedly may be, I should like to draw attention to these two cases as instancing the older view, equally probable, that arteriosclerosis may cause diabetes. The case of Addisonian anæmia, Case XXXIX, is an isolated example of another association which has been recorded recently. Among twenty-five cardiac patients with positive Wassermann reactions it is not surprising to find two suffering from *tabes dorsalis*. Paul D. White's[37] figure for the incidence of syphilis of the central nervous system in patients with cardio-aortic syphilis is 10%. It is interesting that in Case XLIX *tabes dorsalis* was associated with a rheumatic valvular lesion. Cases of cerebral arteriosclerosis, two cases of hemiparesis, cases of retinal vein thrombosis and of femoral thrombosis, cases of Buerger's disease and of migraine emphasize the widespread nature of vascular disease and its two tendencies, organic and spasmodic occlusion. Perhaps it is not going too far to group under this heading the two cases of peptic ulcer. There were also two cases of arthritis, and Cases XVI, XXVIII, XLI, LXXVII, XCVIII and XCIX may serve to show the aggravating influence of sepsis. Unfortunately, detailed attention was not paid to dental sepsis. Case LXXIV among others, however, does show that attention to this factor may bring about amelioration of symptoms, including pain. The interpretation of *herpes zoster* in Case LXXXI is intriguing; should it be regarded as a mere accident or as of closer significance? Case XXVI was interesting because the strain thrown on the myocardium by the accident of gas poisoning was nearly fatal and precipitated a grave disability from which recovery occurred only slowly and in small part. As a contrast, in Case XCIX the septicæmia supervened on a cardiac infarction; yet, after lying at death's door for a month the patient recovered and her systolic blood pressure, having fallen over 100 millimetres of mercury, has now steadily risen again nearly to pre-catastrophic levels.

TABLE X.

Other Diseases Observed in Course of Investigation.

Case Number.	Disease.
II	Chronic nephritis.
IV	*Diabetes mellitus.*
VI	Duodenal ulcer.
IX	Femoral vein thrombosis.
XIII	*Tabes dorsalis.*
XVI	Chronic appendicitis.
XVIII	Osteoarthritis of both knees.
XXVI	Carbon monoxide poisoning.
XXVIII	Cystitis.
XXX	Migraine.
XXXII	Chronic nephritis.
XXXIX	Addisonian anæmia.
XLI	Septic throat, dental sepsis.
XLVI	Rheumatoid arthritis.
XLIX	*Tabes dorsalis.*
LI	Chronic nephritis.
LII	Peptic ulcer.
LXI	Left hemiparesis (old cerebral vascular lesion).
LXIII	Chronic nephritis.
LXXIII	Left hemiparesis (old cerebral thrombosis).
LXXVI	Cerebral arteriosclerosis.
LXXVII	Sinusitis and old phthisis.
LXXXI	*Herpes zoster.*
LXXXII	Central retinal vein thrombosis.
LXXXIII	Peptic ulcer.
LXXXV	*Diabetes mellitus.*
XCIV	Buerger's disease.
XCVII	Concussion.
XCVIII	Hæmorrhoids; cervicitis.
XCIX	Staphylococcal septicæmia (recovery).

Ætiological Groups.

Hypertensive Heart Disease.

Tables XI and XIA show that there were 50% of cases of hypertensive heart disease. Twenty-seven patients were males (54%) and twenty-three were females (46%), with the striking difference that the average age at the onset of pain was twelve

TABLE XI.

Males.

Case Number.	Age at Onset.	Duration of Life Since Onset in Years and Months.
III	59	5 3
V	72	
VIII	59	
XI	65	1 0
XVII	63	0 2
XXIII	57	10 0
XXIX	60	4 0
XXXIII	42	
XXXVII	67	
XL	55	3 6
XLII	41	1 0
XLV	60	3 0
LV	49	2 2
LVII	57	3 0
LVIII	77	1 0
LXV	58	6 0
LXVII	45	3 9
LXIX	43	1 0
LXX	72	0 3
LXXVI	61	2 0
LXXIX	65	0 4
LXXXIV	77	0 8
LXXXV	59	2 0
LXXXVI	81	0 6
LXXXIX	65	1 0
XC	45	2 3
XCIV	54	0 5
Average	.. ..	2 4

TABLE XIA.

Females.

Case Number.	Age at Onset.	Duration of Life Since Onset in Years and Months.
IV	48	17 0
X	50	10 0
XXI	35	5 0
XXVI	53	
XXVIII	58	6 6
XXXI	63	3 0
XXXVI	50	4 0
XLVI	58	3 6
LIII	53	3 0
LIV	47	3 0
LX	54	6 0
LXVIII	52	3 0
LXXIII	51	1 6
LXXIV	46	3 6
LXXV	49	3 6
LXXX	53	2 1
LXXXI	45	2 3
LXXII	42	3 6
LXXXIII	56	0 2
XCIII	39	12 0
XCVI	53	0 8
XCIX	43	3 0
C	34	6 6
Average	.. ..	4 8

years earlier in women (about the period of the climacteric) than in men—in women, may we say, at an age when vascular spasm is more frequent; in men at an age when some degree of the organic sclerosis of advancing years may reasonably be expected. Out of the sixty-seven patients in the series who showed hypertension, sixteen, or 24%, had positive Wassermann reactions. (Lian, quoted by Carey Coombs,[38] reported an incidence of 29%.)

Chronic Rheumatic Carditis.

Of seventeen rheumatic patients, there were ten males (17%), in whom pain on the average first showed itself at the age of thirty-three years, and seven females (17%), in whom it appeared much later, at forty-two years. There were nine patients with mitral disease, five with aortic disease and three with the double lesion. Paul D. White[39] in New England found that the proportion of rheumatic heart lesions (all forms) ranged from 31% to 54%; he also found a predominance of females over males of four to three; and the proportion of mitral, aortic and combined lesions[40] was 62%,

TABLE XII.

Males.

Case Number.	Lesion: Aortic, Mitral.	Age at Onset.	Duration of Life Since Onset of Pain in Years and Months.
VI	A.	39	5 8
XXIV	A. (also C.I.)	56	4 0
XXVII	M.	11	5 6
XXX	A. and M.	43	2 6
XLVII	A. and M.	10	20 0
XLVIII	M.	36	1 8
XLIX	A. (also S.)	52	1 3
LIX	A. and M. (also H.)	34	
LXIV	M.	18	3 0
LXXI	M.	31	0 2
10 cases	Average.. ..	33	4 10

C.I.=Cardiac infarction. S.=Syphilis. H.=Hypertension.

Females.

Case Number.	Lesion: Aortic, Mitral.	Age at Onset.	Duration of Life Since Onset of Pain in Years and Months.
IX	M.	25	
XII	M. (also H.)	25	3 6
XX	A. (also H.)	55	
XXXIX	M. (also H.)	58	5 0
LXII	A.	40	16 0
XCI	M. (also S.)	43	0 4
XCV	M.	37	2 0
7 cases	Average.. ..	42	5 11

C.I.=Cardiac infarction. S.=Syphilis. H.=Hypertension.

5% and 33%. Four patients had hypertension as well, two had positive Wassermann reactions, and one suffered a cardiac infarction. These rheumatic cases account for all the patients in whom pain commenced by the age of thirty years, with the exception of one man who had a cardiac infarction at twenty-six years and a second who had toxic myocardial degeneration at thirty years. It is noteworthy that pain may commence early in rheumatic mitral disease and recur with exacerbations or relapses at intervals. Pain with this lesion is more variable than with any other.

Cardio-Aortic Syphilis.

There were sixteen cases of definite cardio-aortic syphilis, five of aortitis (dilatation shown radiographically in all but one), ten of regurgitation, and four definite aneurysms (all except one of which were associated with regurgitation). The average age of the patients at the onset of pain in this group was in men fifty-five and a half years and in women forty-eight years. It is to be noted that hypertension was present in more than half of these patients, which is somewhat in opposition to the idea generally held that cardio-aortic syphilis is not

TABLE XIII.

Males.

Case Number.	Lesion.	Age at Onset.
XIV	Aneurysm.	60 (also H.).
XV	Regurgitation.	70 (also H.).
XXII	Aneurysm and regurgitation.	60
XXV	Aortitis.	56 (also C.I. X ray+).
XXXV	Regurgitation.	40
XLIII	Aneurysm and regurgitation.	60
XLIV	Regurgitation.	46
L	Aortitis.	49
LII	Aortitis, later regurgitation.	58 (also H. and C.I., X ray +).
LXVI	Aortitis.	54 (also H. X ray +).
LXXII	Aortitis.	61 (also H. X ray −).
XCII	Regurgitation.	52

Females.

Case Number.	Lesion.	Age at Onset.
XIX	Aortitis.	50 (also H.).
XXXIV	Regurgitation.	45 (also H.).
XXXVIII	Regurgitation.	55 (also H.).
LXI	Aneurysm and regurgitation.	43 (also H.).

C.I.=Cardiac infarction. H.=Hypertension. X ray +=X ray findings positive. X ray −=X ray findings normal.

associated with raised blood pressure. Carey Coombs[38] found only one case of cardiac infarction in one hundred and three patients with cardio-aortic syphilis; in this small group of sixteen it occurred twice. Coombs also found 8% of syphilitic cases in a series of *post mortem* examinations on patients dying from heart disease; but he remarks that syphilis is twice as liable as other conditions to cause pain on effort. The average age in his series of one hundred and three patients was fifty years, as compared with fifty-three years in my group. The sex incidence for the whole series was 21% for males and 9·5% for females.

TABLE XIV.

Hypertensive Readings in Cardiac-Aortic Syphilis.

Case Number.	Age When Seen.	Sex.	Systolic Blood Pressure in Millimetres of Mercury.
XIV	60	M.	165
XV	70	M.	200
XIX	52	F.	145 (after C.I.)
XXXIV	45	F.	160
XXXVIII	55	F.	180
LII	64	M.	175
LXI	43	F.	156
LXVI	64	M.	160
LXXII	62	M.	168
Average ..	57		168

Chronic Nephritis.

There is a small group of four patients, two males and two females, with chronic nephritis; although two gave positive Wassermann reactions and one was found to have aortic dilatation on radiographic examination, they are best classed under this heading.

TABLE XV.

Details of Nephritis Patients.

Case Number.	Clinical Findings.
II	With arteriosclerosis, cardiac enlargement, auricular fibrillation and also some aortic dilatation, and very poor renal efficiency and a positive Wassermann reaction. (Male, 75 years of age at onset when first seen. Blood pressure not raised.)
XXXII	Also with enlarged heart, hypertensive, obesity, sclerotic retinal arteries, extrasystoles, several eclamptic pregnancies and three close relatives with Bright's disease and poor renal efficiency. (Female, 45 years of age when first seen; 38 years of age at onset.)
LI	Enlarged heart, hypertension; very poor renal efficiency. (Female, 56 years of age when first seen, at onset.)
LXIII	Cardiac dilatation and hypertrophy, albuminuria, hypertension, extrasystoles, positive Wassermann reaction. Poor renal efficiency. (Male, 70 years of age when first seen; 68 years of age at onset.)

Cardiac Infarction.

Benson and Hunter[41] in two hundred *post mortem* examinations showing coronary obstruction found seventy-six cases of infarction. In this series of one hundred patients investigated clinically from the point of view of pain, twenty-eight had infarction. Of these twenty-eight, 25% were women, which tallies closely with Levine's[36] figure. Of twenty-five who were traced at December 31, 1934, 28% had died. The average duration of life since the onset of pain has been just over five years. It will be seen that in twelve cases the onset of infarction appears to have been the first experience of pain; in two no definite catastrophe could be demarcated in a prolonged history of severe pain—the evidence is electrocardiographic. In the remainder the average duration of pain before infarction took place was three and a quarter years, or, if we exclude Case VII, in which the patient gives the unusual history of having experienced pain for nineteen years before the onset of really severe symptoms, the average for all patients is just over one year. Of the twenty-eight, nineteen had hypertension, three had syphilis, one had rheumatic carditis, one *diabetes mellitus*, and one Addisonian anæmia. A few gave no evidence of the underlying disease, but, as they were seen after the occurrence of infarction, the possibility of previous hypertension has to be borne in mind; these are shown in Table XVII.

TABLE XVI.

Cases of Cardiac Infarction.

Case Number.	Sex.	Age at Onset.	Duration of Life Since Onset in Years and Months.	Duration Before Cardiac Infarction in Years and Months.	Present State.	Disease.	Evidence.
III	M.	59	5 3	0 9	A.	H.	E.K.G.
V	M.	72		0 4	U.	H.	B.P. fall.
VII	M.	32	23 0	19 0	A.		B.P. rise.
X	F.	50	10 0	Indef.	A.	H.	E.K.G.
XVI	M.	26	3 6	Nil	A.		Rub.
XVII	M.	63	0 2	Nil	D.		Rub.
XIX	F.	50	3 0	1 0	D.	H.S.	B.P. rise.
XXIII	M.	57	10 0	8 0	D.		B.P. rise.
XXIV	M.	56	4 0	1 0	D.	R.	B.P. rise.
XXV	M.	56	3 6	0 1	A.	S.	Rub.
XXIX	M.	60	4 0	Nil	D.	H.	E.K.G.
XXXIII	M.	42		Nil	U.	H.	Clin.
XLV	M.	60	3 0	Nil	A.	H.	Clin.
LII	M.	58	9 0	2 0	A.	H.S.	E.K.G.
LIII	F.	53	3 0	Nil	A.	H.S.	Clin.
LV	M.	49	2 2	Nil	A.	H.	Clin.
LVI	F.	45		Nil	U.		Clin.
LVII	M.	57	3 0	Nil	A.	H.	B.P. rise.
LXIII	M.	68		2 0		H.N.	Clin.
LXV	M.	58	6 0	0 5	D.	H.	E.K.G.
LXVII	M.	45	3 9	Nil	A.	H.	Clin.
LXVIII	F.	52	3 0	2 6	D.	H.	E.K.G.
LXXVIII	M.	45	9 6	Nil	A.		Clin.
LXXIX	M.	65	0 4	Nil	A.	H.	Clin.
LXXXV	M.	59	2 0	1 3	A.	H.D.	E.K.G.
LXXXVI	M.	81	0 6	Indef.	A.	H.An.	E.K.G.
XCIX	F.	43	3 0	2 0	A.	H.	E.K.G.
C	F.	34	6 6	5 3	A.	H.	Clin.

M. = male; F. = female; A. = alive; U. = unknown; D. = dead; H. = hypertensive heart disease; R. = chronic rheumatic carditis; N. = nephritis; D. = *diabetes mellitus*; An. = Addison's anæmia; E.K.G. = electrocardiogram; B.P. = blood pressure; Clin. = clinical evidence; Rub = pericardial friction rub; S. = syphilis.

TABLE XVII.

Case Number.	Sex.	Clinical Findings.
VII	M.	Seen after infarction; heart enlarged; pressure has risen slightly since.
XVI	M.	Cardiac infarction in young man healthy except for dental sepsis and chronic appendicitis.
LVI	F.	Seen after infarction with low pressure; cause doubtful.
LXXVIII	M.	Seen after infarction with low pressure; cause doubtful.

Toxic Myocardial Degeneration.

Apart from the foregoing groups of hypertensive heart disease, chronic rheumatic carditis, cardio-aortic syphilis, chronic nephritis and cardiac infarction, there remain nine cases, further details of which are set out in Table XVIII. It will be observed that there is no doubt about the presence of an organic cardiac lesion, perhaps with the exception of Case XLI, the only case in the group from which cardiac enlargement is absent. What is equally striking is: the high incidence of cholecystitis and of spondylitis, both present in seven out of the nine cases; the presence of sepsis in more than half; and the blood pressure raised slightly in one and temporarily raised in one other, leaving the majority without this feature which is so frequent in the series as a whole. This miscellaneous group of cases, left by a process of exclusion, may perhaps suitably be described as due to toxic myocardial degeneration.

TABLE XVIII.

Case Number.	Sex.	Age when First Seen.	Clinical Findings.
I	F.	59	Breathlessness, cardiac asthma, œdema, enlarged heart, cholecystitis, spondylitis, low blood pressure.
XIII	M.	51	F.H.+, syphilis, œdema, breathlessness, enlarged heart, cholecystitis, spondylitis, poor renal function, low blood pressure.
XVIII	F.	55	Breathlessness, cardiac asthma, œdema, enlarged heart, spondylitis, low blood pressure, myxœdema.
XLI	M.	36	Septic throat, gross dental sepsis, arteriosclerosis, loss of weight, cholecystitis, spondylitis, old rheumatic history, hypertension (temporary), E.K.G. physiological.
LXXVII	M.	50	F.H.+, arteriosclerosis, hypertension (slight), old phthisis, spondylitis, cholecystitis, enlarged heart, breathlessness, tachycardia, sinusitis, E.K.G., left axis deviation.
LXXXVII	F.	40	F.H.+, breathlessness, œdema, tachycardia, low blood pressure, cholecystitis, dental sepsis, cramps in calves, cardiac enlargement, E.K.G., myocardial degeneration.
LXXXVIII	F.	43	F.H.+, enlarged heart, low blood pressure, cholecystitis, gross pyorrhœa, spondylitis, breathlessness, E.K.G., myocardial degeneration.
XCVII	M.	77	Spondylitis, no elevation of blood pressure, very slight breathlessness, E.K.G., myocardial degeneration, severe cramps, emphysema masks cardiac dulness, but heart almost certainly enlarged.
XCVIII	F.	37	F.H.+, tachycardia, breathlessness, cholecystitis, blood pressure not raised, E.K.G., myocardial degeneration, enlarged heart, uterine sepsis, history of rheumatic carditis.
Average age	..	50	

F.H.+=Family history gives evidence of relevant disease.

Summary of Ætiological Groups According to Sex.

It will be seen from Table XIX that the incidence of disease is different in the sexes. Hypertensive heart disease heads the list and, with myocardial degeneration, is more frequent in women. Chronic rheumatic carditis and nephritis have about the same sex incidence. Syphilis is twice as common in men as in women, and so is cardiac infarction of doubtful origin.

TABLE XIX.

Disease.	Males.		Females.	
	Total.	Percentage.	Total.	Percentage.
Hypertension	27	47	23	55
Syphilis	12	21	4	10
Rheumatic	10	17	7	17
Myocardial degeneration	4	7	5	12
Cardiac infarction	3	5	1	2
Chronic nephritis	2	3	2	4
Total	58		42	

Deaths.

We come now to a consideration of those seventeen patients who are known to have died; they are set out in Table XX. It will be observed that only seven, or less than half, gave evidence of cardiac infarction; five were rheumatic subjects; three suffered from uncomplicated hypertensive heart disease; four had positive Wassermann reactions. Only three were women; one of these had syphilitic aortic regurgitation; one had hypertensive heart disease and cardiac infarction; and the third had hypertension, syphilis and infarction. The prognosis

TABLE XX.

Case Number.	Sex.	Age at Onset.	Age at Death.	Duration of Pain in Years and Months.	Cause.	Mode.	Remarks.
VI	M.	39	44½	5 6	Chronic rheumatic carditis.	Sudden in congestive.	P.M.N.G.
XI	M.	65	66	1 0	Hypertension.	Congestive.	P.
XV	M.	70	74	4 0	Syphilis, aortic regurgitation, hypertension.	Unknown.	P.
XVII	M.	63	63	0 2	Infarction.	Suddenly.	P.M.N.D.
XIX	F.	50	53	3 0	Infarction, syphilis, hypertension.	Congestive.	P.
XXIII	M.	57	67	10 0	Infarction.	Congestive.	P.
XXIV	M.	56	60	4 0	Chronic rheumatic carditis.	Dyspnœic.	P.M.N.D.
XXIX	M.	60	64	4 0	Infarction, hypertension.	Congestive.	P.
XXX	M.	43	45½	2 6	Chronic rheumatic carditis.	Unknown.	P.
XXXIV	F.	45	45	0 2	Syphilis, aortic regurgitation.	Congestive.	P.M.N.D.
XXXV	M.	40	41	1 2	Syphilis, aortic regurgitation.	Sudden, acute pain, cyanosis.	P.M.
XLII	M.	41	42	1 0	Hypertension.	Dyspnœic.	P.M.
XLVII	M.	10	30	20 0	Chronic rheumatic carditis.	Sudden in congestive.	P.M.
XLIX	M.	52	53	1 3	Chronic rheumatic carditis.	Sudden.	P.
LVIII	M.	77	78	1 0	Hypertension.	Uræmic.	P.M.
LXV	M.	58	64	6 0	Infarction.	Sudden.	P.
LXVIII	F.	52	55	3 0	Infarction, hypertension.	Sudden, acute pain.	P.

P.=Died outside hospital.
P.M.=*Post mortem* examination done.
P.M.N.D.=Died in hospital, *post mortem* examination not done.
P.M.N.G.=Died in hospital, *post mortem* examination not granted.

in women, therefore, seems definitely better than in men, possibly owing to the greater part played by functional lesions in women. The average duration of life from the onset of pain in the fatal cases is the same as the average for the series as a whole.

Average Duration of Life since Onset of Pain, in Ætiological Groups.

Of the eighty-two patients who were within knowledge at the end of December, 1934, seventeen have died, or nearly 21%. The duration of life ranges from twenty years in a patient with chronic rheumatic carditis to the two months by which a man who ultimately died of cardiac infarction survived his first experience of pain; the average duration since the onset of pain was 4·4 years. L. E. Viko,[42] investigating a series of one hundred and fifty-three patients diagnosed as having coronary sclerosis, found the average length of life from the onset of symptoms to be 4·3 years. The duration since the onset of pain is analysed according to sex and disease in Table XXI. It will be seen that, while the outlook is roughly the same in both sexes in chronic rheumatic carditis, in myocardial degeneration and in cases which proceed to cardiac infarction, in hypertensive heart disease the prognosis is far worse in men; in cardio-aortic syphilis the reverse is true. It will be noticed that the small group of nephritis cases is not included; unfortunately not one of these could be traced at December 31, 1934—presumably they have all died. (With regard to the eighteen patients in all who could not be traced, it should be explained that most of the series are of the public hospital class; bitter experience shows that they are a nomad race, with whom all entreaties to keep one advised of any change of address are likely to be unavailing.)

TABLE XXI.

Duration of Life Since Onset of Pain.

Disease.	Males. Years and Months.	Females. Years and Months.
Hypertensive heart disease	2 4	4 8
Cardio-aortic syphilis	5 0	2 1
Chronic rheumatic carditis	4 10	5 11
Myocardial degeneration	3 9	3 11
Cardiac infarction (all cases)	5 2	4 1

Other Aspects.

Under the heading "other aspects" are two subheadings: A, response to pain—motionless or restless; B, clinical findings in cases not showing cardiac enlargement. These two aspects are summarized in Tables XXII and XXIII. It will be seen from the first that no correlation can be established between lesion and response; the second is included in support of the statement made earlier, that definite organic disease was universally present in the series.

One other point which is not shown, but which presented itself very definitely in dealing with these patients, is that when they suffer from ailments causing pain in adjacent neighbourhoods they have no hesitation in differentiating this from their pain of cardiac origin. They are quite sure that the pains arising from peptic ulcer or from cholecystitis or from *herpes zoster* are distinct, though they may, of course, proffer mistaken explanations. It should be emphasized that the failure to record cases of simple neuro-circulatory asthenia means that they were not seen, not that they were discarded. Corresponding statistics have been quoted whenever possible to show that the present series is a fairly comprehensive one in comparison with the findings of other workers, allowance being made for its size and the differences of race and climate.

TABLE XXII.

Case Number.	Sex.	Patient's Description.	Disease.
I	F.	Has to stand still.	Myocardial degeneration.
III	M.	Stops.	—
V	M.	Stops.	—
VI	M.	Stops.	Chronic rheumatic carditis, aortic stenosis.
XI	M.	Made him rest.	Hypertension.
XIII	M.	He stops and rubs it away.	Myocardial degeneration.
XVI	M.	Rubbed it . . . had to keep walking about.	Cardiac infarction.
XVII	M.	Stood still.	Cardiac infarction.
XXI	F.	Relieved by rest.	Hypertension.
XXII	M.	He has to stop.	Aneurysm.
XXIII	M.	Had to stop.	Cardiac infarction.
XXV	M.	Just wants to grasp his chest and stand still.	Cardiac infarction.
XXVI	F.	Feels breast needs support.	Hypertension.
XXVII	M.	Has to sit down and hold his breath.	Chronic rheumatic carditis.
XXVIII	F.	She had to sit down and hold herself.	
XXX	M.	He has to sit and rest completely.	
XXXI	F.	Has to press on breast while walking.	Hypertension.
XXXII	F.	You've got to keep still.	Hypertension, nephritis.
XXXIII	M.	He stood . . . pressing the painful area.	Cardiac infarction.
XXXIV	F.	Couldn't get her breath, but had to walk about.	Syphilis, aortic regurgitation.
XXXV	M.	He couldn't have gone on.	Syphilis, aortic regurgitation.
XXXVIII	F.	Rubs chest for relief.	Syphilis, aortic regurgitation and hypertension.
XL	M.	Had to sit down. Partly relieved by rubbing.	Hypertension.
XLVIII	M.	Had to stop dead.	Chronic rheumatic carditis.
LV	M.	Had to pull up.	
LVII	M.	He was unable to move.	Cardiac infarction.
LX	F.	Had to lie still with it.	Hypertension.
LXII	F.	Makes her lie down.	Chronic rheumatic carditis.
LXVI	M.	Tosses about all night.	Syphilis, aortitis, hypertension.
LXIX	M.	He had to stop.	Hypertension.
LXX	M.	He keeps still when it comes on.	Hypertensive heart disease.
LXXVIII	M.	Rolled about in agony.	Cardiac infarction.
LXXIX	M.	Had to stop.	Cardiac infarction, hypertension.
LXXX	F.	She has to pull up.	Hypertension.
LXXXIII	F.	Has to pull up.	Hypertension.
LXXXIV	M.	Has to keep still.	Aortic aneurysm and regurgitation.
LXXXV	M.	Had to stop.	Cardiac infarction.
LXXXVII	F.	Has to hold the left breast. Can't move . . .	
LXXXVIII	F.	Had to lie still, couldn't stand nor sit.	
XCIV	M.	Had to sit down.	Hypertension.
XCVI	F.	Has to stand still.	Hypertension.
XCVIII	F.	Has to sit down.	Myocardial degeneration.
XCIX	F.	Has to stop.	Hypertension, cardiac infarction.
C	F.	Massaged left arm for relief.	Hypertension, cardiac infarction.

Eleven resort to pressure or massage; thirty-seven are impelled to keep still; four toss or roll or walk about (two syphilis and cardiac infarction). All three reactions are to be seen in cardiac infarction in Cases XVI, XXXIII and LXXVIII; in syphilis in Cases XXXIV, XXXV and XXXVIII; in hypertensive heart disease in Cases LX and XL and in chronic rheumatic carditis in Case XXXIX.

TABLE XXIII.

Cases not showing Cardiac Enlargement.

Case Number.	Sex.	Clinical Findings.
XII	F.	Early rheumatic mitral lesion, hypertension (early).
XXXVII	M.	Hypertensive heart disease, sclerosed radial arteries, calcified aorta.
XLI	M.	Temporary hypertension, old rheumatic history, extensive focal sepsis.
XLVI	F.	Hypertension, septic tonsils, diseased gall-bladder, multiple arthritis.
XCIII	F.	Hypertension, cholecystitis, obesity (obscures).
XCIV	M.	Hypertension, deformed chest.
XCVIII	F.	Cardiac infarction, cholecystitis, tachycardia.

We come now to a review of the general picture presented by patients complaining of cardiac pain, as represented by this series of one hundred unselected cases. In more than half there is a history of cardiac disease in immediate relatives. If the patient is a man, which is a little more likely, he will be about fifty-four and a half years old; and if a woman, she will be about fifty. Half the patients have hypertensive heart disease; this is somewhat more frequent in women, and in them pain appears earlier than in men; in both it appears on the average at what might be considered the age of the climacteric. The longer the duration of pain, the more likely is it to be associated with high blood pressure. The next group in point of size is that of the patients with chronic rheumatic carditis, which occurs in association with pain with equal frequency in men and women, though, unlike hypertensive heart disease, it occurs at an earlier age in men. Almost as common is cardio-aortic syphilis, which is twice as frequent in association with pain in men, in whom it occurs at a later age. Following on these there is a small group in which advanced chronic nephritis is the chief feature, roughly of equal distribution. Finally there is a miscellaneous group, classified as myocardial degeneration, which also occurs later in men and which is almost constantly associated with spinal and gall-bladder disease.

Cardiac infarction occurs in 28% of cases and is three times as frequent in men. It is most commonly found in association with hypertension, occasionally with syphilis, and rarely with chronic rheumatic carditis. The occurrence of non-fatal cardiac infarction makes little difference to the ultimate duration of life from the onset of pain. It is interesting to note that in half the patients with cardiac infarction pain had been present before the catastrophe. Gall-bladder disease and spondylitis are notably associated with pain in cardiac disease—as commonly as is infarction—not only in myocardial degeneration. The significance of these has been discussed under the appropriate headings.

The pain itself presents a varied picture. In about half the cases it is the first symptom complained of; in nearly all the remainder it develops subsequently to the onset of breathlessness; but almost universally the two become associated before long; and in not a few the disappearance of pain heralds a fatal termination. Sometimes its duration is a matter of minutes only or even seconds, almost as often of hours, and less frequently of days. But the duration of the pain is no guide to the nature or severity or prognosis of the underlying lesion. Its sites are more varied than its durations, and it is highly unusual for any one patient to experience pain in one site only throughout the course of his illness. While the pain is mostly either about the mid-line or left-sided, it can occur on the right side, and ranges from the upper cervical to the lower dorsal level of the spinal nerves. Both its onset and cessation may be brief or gradual, and, while it may occasionally be induced by cold or emotion or may appear entirely unexpectedly, in almost all cases it is produced or aggravated by exertion. Radiation is a common feature, as is hyperalgesia; and another association is cramps in the lower extremities, more frequently found in men. On examination definite cardiac enlargement was almost universal, and electrocardiographic changes were present in the great majority. As to prognosis, the average duration of life after the onset of pain was 4·4 years; in the several groups the figures are: rheumatic, 5·0; myocardial degenerative, 4·0; hypertensive, 3·5; syphilitic, 3·0. Except in syphilitic cases, the prognosis is distinctly better in women.

From this survey cardiac pain would appear: to be almost invariably associated with definite, detectable organic heart disease and, further, with most of the main types of organic disease; to be frequently associated with evidence of arterial disease elsewhere; not to be characteristically sternal in site; rarely to be associated with *angor animi*; and, on the average, to be of grave prognostic significance, taken by itself regardless of its context. It must be admitted that this agrees wholly neither with Heberden's original description of *angina pectoris* nor with many modern views on cardiac pain. Before proceeding to an attempt at classification, some effort should be made to review current ideas on the causation of this pain in the light of the above findings. Any such explanation must take into account: the varied nature of its onset and cessation, of its duration, of its position, of the underlying disease; certain of the associated features commonly found; and the nature of precipitating factors.

The main points in cardiac anatomy and physiology which concern us are as follow. It is known that the walls of the coronary arteries contain muscle fibres, and that these can both contract and dilate has been shown on many occasions and in various ways. Woollard[43] has shown that there is a rich innervation of the coronaries; and Anrep and Segall[44] have shown that the vagus has a constrictor and the sympathetic a dilator effect. Laubry, Walser and Deglaude[45] demonstrated both dilatation and constriction with nicotine in varying strengths, and Fenn and Gilbert[46] suggest that digitalis may bring about coronary constriction, while Brunton[47] states that both digitalis and theobromine have a dilator effect. (In Case VIII it was clearly shown that large doses, twenty minims

four times a day, aggravated pain, while smaller doses, ten minims taken three times a day, definitely relieved it.) It has been shown that there are pain fibres running from the heart by way of the cervical sympathetic trunks and the upper thoracic ganglia to the upper dorsal posterior nerve roots, especially from the eighth cervical to the fourth dorsal (Samson Wright,[48] John Cowan[49] and Lambert[50]). For the moment the cardio-accelerator and cardio-inhibitory reflexes, so well summarized by FitzGerald Peel,[51] do not concern us so much.

Many hypotheses and suggestions have been advanced as to the causation of pain. Allan Burns in 1806 suggested a coronary origin; Allbutt[1] is the most noted protagonist of the aortic school; neuritis of cardiac and aortic nerves was observed histologically by Lancereaux,[52] and Romberg[52] suggested a neuralgia. Cramp of the heart is a facile notion which would explain much, but which lacks supporting evidence; and recently Sir Thomas Lewis,[15] in his St. Cyres Lectures, has put forward the idea of the formation and accumulation of a pain-producing factor.

Of one set of circumstances there can be no doubt. In cardiac infarction due to coronary embolism there is sudden agonizing pain, of long duration, gradually subsiding, but not infrequently leaving a tendency to similar pain of less severity on exertion. This pain we know is associated with a sudden block of the vessel lumen and a corresponding area of infarction. Almost similar events are seen in cases of coronary thrombosis in the present series, for example, Cases XVI and XVII. Such a pain may possibly be due to the stimulus of anoxæmia, or of Lewis's "*p*" substance, or of a shortage of glucose[53][54][55] on the nerve endings so richly scattered throughout the myocardium.[56][57] But this explanation will not suffice for cases such as XXII and XLII, in which attacks of pain of briefer duration take place repeatedly, and in which there can be no such permanent obstruction. With all respect to Allbutt, we now know definitely that coronary thrombosis is not an aortic phenomenon; and against the aortic origin of these other pains we have to weigh the ever-increasing bulk of evidence that there are repeated paroxysms associated with electrocardiographic changes, closely similar to those characteristic of coronary thrombosis. Gray[58] reports electrocardiographic changes in paroxysms of pain similar to those of definite infarction. Hamilton and Robertson[59] report similar changes in electrocardiograms taken *in articulo mortis* associated with pain in a patient, *post mortem* examination of whom revealed coronary sclerosis but no infarction. Dietrich and Schwiegk[60] found that electrocardiographic changes similar to those associated with pain could be produced experimentally by relative disproportion between coronary flow and work demands on the myocardium, either by constricting the coronaries or by decreasing the supply of available oxygen. This evidence goes to show strongly that in many cases without organic obstruction changes are produced, precisely similar to those of the obstruction of infarction, by relative alteration of the coronary flow. It may also clear up the difficulty previously felt in explaining pain by ischæmia in the presence of hypertrophy. Further support to the idea of relative values is lent by cases of pain occurring in paroxysmal tachycardia; there was one in this series (Case XI), and several have been reported by Wolff,[61] Hall,[62] and White and Camp.[63]

While there is no real evidence for myocardial cramp, there is much to support the view that localized vaso-constriction of the coronary arteries may occur. These arteries have muscle under nervous control, which, we have seen, is exercised. Similar events do occur elsewhere, and in extremely localized fashion, as Lewis[64] has shown in the fingers in Raynaud's disease; and he even goes further to suggest that the primary cause may be local. That such events take place is equally well known in Buerger's disease, in which there is definite disease of the arterial wall. The significance of this is well discussed in a different connexion by Telford and Stopford,[65] whose remarks are of value for cardiology as well.

> In the disease at present known as *thrombo-angiitis obliterans* there is a combination of vaso-spasm with arterial thrombosis . . . vaso-spasm is the initial lesion, and prolonged, severe vaso-spasm induces thrombosis by interference with the blood supply to the vessel wall . . . It is necessary in the investigation of all cases of vascular disease to determine, if possible, the extent of the vaso-spasm which is superimposed on the completely or partially occluded vessels.

If Buerger's disease has done nothing else, it has been of great service in dispelling the preconception that organic disease of vessel walls and spasm are mutually incompatible. Nordmann[66] showed that structural changes in the aorta were proportionate to the hypertension present; and in an X ray study of the coronary vessels *post mortem* it was shown that commencing coronary sclerosis is almost universal after the age of thirty years. Of the occurrence of localized vascular spasm I myself have ocular evidence in a case, as yet unpublished, of *amaurosis fugax*,[67] which Hughlings Jackson has also observed. Granted relative insufficiency of coronary flow as an adequate stimulus,[68] coronary angiospasm will explain much or most of the variations of cardiac pain in mode of onset and cessation, duration, severity and site. Attention is drawn to Table X, in which the frequency of other vascular disease has been recorded. The views of Telford and Stopford, already quoted,[65] and of Telford,[69] who describes the development of organic vascular changes following on sympathetic over-action due to irritation by cervical rib, explain the march of events in such cases as XXIII; and they give us a basis of explanation for the rheumatic cases with pain in early life, the pathological changes in which have recently been recorded by Karsner and Bayless.[70] Bizarre examples to lend support to this coronary view are the occurrence of pain, first in *periarteritis nodosa* of the coronaries,[71] and

secondly in a case reported by Eppinger[72] in which coronary occlusion followed dragging on the vessel by an adhesion connected to a diaphragmatic hernia. Spasm, leading to ventricular fibrillation and death, as Lewis has shown experimentally by ligation, may be accepted as the most likely cause in those fatal cases which have been reported as not showing complete coronary obliteration. That in small vessels minute infarctions may occur must be admitted—in Raynaud's disease Lewis[64] has shown that small areas of necrosis may be seen histologically, while massive necrosis is unusual.

The adoption of the idea of coronary angiospasm leads to another possibility as regards pain, especially its sudden onset. During my association as a house surgeon with Dr. C. E. Corlette at Sydney Hospital, I observed time and again that, after extensive local infiltration with an anæsthetic solution, otherwise completely efficient, application of pressure forceps to vessel walls caused appreciable pain. Waterston,[73] experimenting with acupuncture, found that arteries gave a very acute response of immediate, severe and sickening pain, which tended to radiate along the course of the vessel. Pickering and Hess,[74] while working with histamine, reported evidence to show that headache is due to stretching of the walls of the cerebral arteries or of the tissue in their immediate neighbourhood. Indirectly this is borne out by the experiments of Leriche,[75] who found that, while ligation of the coronary vessels produced ventricular fibrillation, anæsthetization of the adventitia counteracted it; this result is also to be seen in the work of Sutton and Lueth.[76] Perlow, Markle and Katz[77] found that stasis and anoxæmia were also important in delaying the abatement of pain, which reminds us of such cases as Case XXII, in which the pain, having come on acutely, dies away gradually. On these grounds it is my belief, a view supported by Grant,[78] that cardiac pain not due to infarction is dependent on relative coronary insufficiency, caused by increased work and/or coronary angiospasm. This explanation is adequate for all the variations of pain shown in the series, whether the biochemical changes be hypoglycæmic, anoxæmic, or excess of Lewis's *"p"* factor, if we consider in these cases without organic block the local effect of vaso-constriction on the nerves in the vessel wall, and also in cases of organic block the effect not only of constriction, but of dilatation. There remains what has been well termed the lancinating type of pain, pains lasting not even for minutes, but only for seconds, and of all grades of severity. These certainly cannot be due to biochemical changes, such as the accumulation of Lewis's *"p"* factor. They present a difficult problem, as do the lightning pains of *tabes dorsalis*, which it has always been hard to understand in such an essentially chronic malady. I am inclined to locate their origin in the spinal nerve roots, where vascular spasm may occur in close association with neurones; though it is also possible that similar conditions may exist with the ganglion cells in the coronary walls. (That such pains may be associated with serious disease I have reason to remember from a patient under my care, who was supposed by her family to be neurotic. She never had any cardiac pain, except of the lancinating type; but she had a definitely enlarged heart, ringing aortic second sound and raised pressure; and she died suddenly—so far as I was concerned at the time, quite unexpectedly.)

We come now to the consideration of the site of the pain, which is connected with the problems of referred pain, radiation and habit. In cardiac pain we have to deal largely with the referred type. Cowan[79] has an excellent diagram, showing that its most frequent sites lie within the area supplied by the spinal segments directly connected with the cardiac nerve supply. Hirschfelder[80] goes further and gives a diagram localizing pain from the four chambers of the heart to the four quadrants of the anterior aspect of the thorax. I must confess that my findings lend little support to this. The marked preponderance of left-sided pain clinically cannot be correlated with the increasing weight of evidence pathologically that coronary disease is not preponderantly left-sided. Referred pain is well described by Foster Moore;[81] in connexion with this it is interesting to note the association of pain and spondylitis in Table IX. There are other views on referred pain, more recently that advanced by Morley,[16] with regard to abdominal pain. But, although Leriche[82] has demonstrated the sensitivity of the pericardia, epicardia and endocardia, and has shown that their stimulation leads to a depression reflex, which he suggests may occasion some sudden deaths, it is difficult to explain cardiac pain along the lines of occasional changes in the surface of the heart impinging on the pericardium. It can, however, be argued that pain behind the upper, middle and lower parts of the sternum, pain in the left side of the neck, along the costal margin and in the left arm and/or hand is associated with different spinal levels. While spread from one to another may be explained by radiation, their isolated occurrence surely implies different sites for their responsible lesions—even more so when the site changes from time to time in the one patient, or when the one patient has two types of pain at once as regards rest and exertion. All this provides an irresistible reminder of Lewis's experiments in Raynaud's disease, in which he was able to produce vaso-spasm at very localized and varying points. This is but a general view, but surely the sooner the term precordial is discarded, the sooner the location of pain is recorded in detail and an attempt is made, when it occurs in isolated sites, to correlate these with electrocardiographic and *post mortem* changes, the sooner we shall arrive at a stage where pain will have a more precise localizing significance for the underlying lesion, just as even now careful consideration of the clinical context has for its pathological nature. Once we accept the conception of relative coronary insufficiency together with the possibilities of pain and spasm in arterial

walls, and when we remember the close connexion of the nerve supplies of the heart and aorta (Daniélopolu[83] indeed insists on calling them cardio-aortic), it is easily seen that pain can be explained along the same lines, whether we have: a rare case of Allbutt's with diseased aortic wall and sensitive nerve endings; coronary insufficiency produced by syphilitic obstruction at the mouths of the coronary arteries, by atheroma anywhere along their course, or by vaso-spasm (associated or not with such diseases as atheroma, rheumatic inflammation, periarteritis, adhesions, and so on); or insufficient flow in lesions of the aortic valves or myocardium.

It will be seen that the above findings do not agree with several commonly expressed opinions. Hall[62] writes:

> Pain of true cardiac origin was of two types: *angina pectoris*, brief and induced by effort, and the prolonged pain of coronary thrombosis usually occurring at rest. The site, always in or near the mid-line, is identical.

This would fail to include many cases in the present series, in which examples to the contrary can be found for every point. Pain in coronary thrombosis is not always near the mid-line;[84] it is not always prolonged; and there are many other pains, truly associated with heart disease, which are of long duration and which occur without effort. As regards radiation, Riesman and Harris[85] state:

> In coronary thrombosis the pain begins during rest and may not radiate, whereas in angina it starts during an effort and radiates. Dyspnœa is marked in thrombosis and rare in angina.

Cardiac infarction pain does radiate; and what is the pain, so frequently associated with dyspnœa in the majority of this series, if not cardiac? The effort bogy is raised by Wedd,[86] who, criticizing a too frequent modern diagnosis of cardiac infarction, holds that substernal or epigastric pain is related to effort if it is of cardiac or aortic origin. While the heart beats, there is always effort, and the importance of relative values is well exemplified by the undoubted effect on such patients of barometric changes. These critical features are based on shibboleths from the past and are wrongly chosen.

Leaving out of consideration patients who are insensitive to pain,[10][87] we are left with the groups so often described as neuro-circulatory asthenia and Gower's vaso-vagal syndrome, which has been attracting much attention recently. In vaso-vagal attacks Ryle[88] stresses the presence of an elaborate subjective picture, with many sympathetic manifestations and vasomotor phenomena; he also emphasizes most importantly the difference between a "sense of dying" and the fear of death which was Heberden's angor. I have endeavoured to discriminate between these, and they are well exemplified in Cases XXXI and XXVIII respectively. Ryle further says that the pain and its radiation and the sense of constriction are similar to those in organic heart disease; and in another place[89] he suggests that vagal and vasomotor phenomena play an important part, which, according to the view put forward above, would be by way of the coronary vessels, either directly or relatively. Lewis[90] discusses Nothnagel's original cases of *angina vasomotoria*, in none of which was there organic heart disease, and refers to the production of similar symptoms in healthy subjects by adrenaline. He agrees that vaso-vagal manifestations may be superimposed on organic lesions, but states that in uncomplicated attacks, which primarily affect young subjects, he has never seen cardiac pain, constriction nor angor.

We come finally to the subject of neuro-circulatory asthenia or effort syndrome. To Willius's[91] view, that its origin is primarily psychological, depending on the universal conception of the heart as the hub of physical and spiritual activity, I cannot subscribe. His "synthetic emotionalism of the arts" might account for submammary and mammary pain, but it cannot reasonably be expected to explain the numerous other sites. Table V has shown that pure mammary pain was rare and always associated with organic heart disease; and K. Shirley Smith[92] quotes a series of cases in which left submammary pain was an almost constant feature of congestive failure. I do not believe that a patient consciously or subconsciously says to himself: "I have, or should like to pretend to have, a diseased heart. In such case I should have pain associated with my heart; therefore I should have a pain here—in fact I have a pain here." As Volborth[93] showed in puppies, when they have never eaten meat they do not salivate when it is presented to them for the first time; though, having partaken, they may do so ever afterwards. K. Shirley Smith[94] mentions three groups simulating heart disease—the effort syndrome, left mammary pain and muscular retrosternal pain—and bases the diagnosis partly upon absence of signs of cardio-vascular disease. Much the same might be said of pain in herpes, ruptured ulcer, cholecystitis and pancreatic lithiasis.[94] There is, however, no reason why a temporary, functional, cardiac disorder, producing pain in an orthodox way, as outlined above, may not be magnified and/or perpetuated by neurasthenic or psychasthenic processes. Gowers[95] said long ago about neural impulses:

> . . . wherever it occurs; wherever it spreads—in every place the discharge leaves behind it a residual state disposing to its repetition.

And in Head's[96] opinion:

> . . . occasionally, central resistance to potentially disagreeable influences is so low—temperamentally—that pain may appear without any obvious cause or peripheral stimulation.

Cassidy[97] writes:

> . . . an apprehensive man who suffers from *angina pectoris* . . . may through sheer funk drift into such a state of vasomotor irritability that anginal attacks are induced on the slightest exertion or emotional upset—another example of a functional superstructure on an organic basis.

Good examples in the present series are Cases VIII and XXXI. It is not usually argued that, because we can find no cause for an epileptic fit,

it is an imitation of neurological disease; functional rather than organic perhaps—but that is not the same thing. Smith[92] would explain such cases by sensitization of the afferent neural paths by some unknown toxic, endocrine or metabolic agent, so that a trivial stimulus is greatly exaggerated; and this I am prepared to accept. The mechanism is the same whether the alteration occurs as an increased stimulus or as a lowered threshold.

And so we come once more to the consideration of the problem which prompted the investigation—a classification of cardiac pain. Detailed analysis of the series has shown that the underlying disease may be representative of most of the types of organic disease affecting the heart. It has shown that the characteristics of the pain may vary greatly as regards site, duration, intensity and mode of onset, while retaining almost universally the associated features of dyspnœa and evidence of organic disease. It has been argued that a coronary explanation of one type and/or another will suffice to explain these variations; and it has been put forward that an extension along similar lines will cover cases of pain in functional disease, that is to say, pain characteristic of a cardiac origin in the absence of evidence of organic disease. It is further to be noted that a majority of the cases investigated fail to come under the heading of definite cardiac infarction; and that still fewer measure up to the canons of *angina pectoris* as laid down by Heberden. It has been shown that sundry recent definitions are far from all-embracing. Silberberg[98] divides *angina pectoris* into: A, four types; B, four types; and C, various combinations of A and B. From another angle, Cotton[22] states: ". . . the distinctive character of the pain of *angina pectoris* is that it can always be produced by effort", whereby 97% of the cases in this series take one step at least towards qualifying; but again: ". . . it is paroxysmal", which would exclude many of them. Are we to hold that pain of long duration, even in the presence of an organic lesion, is neurasthenic? When we find an attempt to establish the pain of anginal type in paroxysmal tachycardias as yet another group[61]—perhaps as "the angina of tachycardia"—we are only passing from confusion to chaos. Parkinson[99] has gone part of the way in pressing for the acceptance of ". . . the essential unity of *angina pectoris* and a coronary thrombosis". Professor Plesch[100] has gone further, though at first sight it might appear in an opposite direction, when he remarks that, so long as all angina and coronary troubles are regarded as the same, not much progress can be expected.

It is my view that the difficulty has arisen from the cardinal error of regarding pain as anything but a symptom. Granted perhaps that no other pain may be so impressive as pain of cardiac origin, in essentials what is *angina pectoris*, even as Heberden described it, but pain? It is hard to depart from the hallowed past, but I quote the words of a leading article in *The British Medical Journal*:[101]

> Although a certain historical value attaches to old views and old terminology, definite harm may accrue from their preservation. Thus, if a certain phrase implies the truth of conceptions which we now know to be false, phraseology must be adjusted or progress must be delayed.

and of Kinnier Wilson:[102]

> . . . if the exact pathogenesis in certain patients who exhibit this symptom cannot be elucidated, ignorance should not be cloaked by the elevation of a symptom into a disease entity.

With these words as support, this thesis draws to its end with the conclusion that there is no such disease entity as *angina pectoris*; that the term should be scrapped; that all pain in heart disease (mild or severe, twinge, paroxysm or prolonged, associated with coronary block, organic or spasmodic, purely functional or erected on an organic basis, even rare cases with Heberden characteristics and negative *post mortem* findings) should be known simply as cardiac pain. This pain may or may not be associated with angina, a sense of constriction, just as angina may or may not be associated with pain. This should no more be a conclusion than a diagnosis of renal colic. As in the latter condition we proceed to a search for the nature of the obstruction, whether stone, kink or external pressure, so the finding of cardiac pain should urge us on to a search for its cause, whether irritation of the nerve endings in an inflamed aorta, coronary embolism or thrombosis or vasospasm, relative inefficiency of coronary circulation, or the perpetuation of an unhappy event by a disordered nervous system. So will our exact diagnosis, and consequent treatment, of cardiac ailments progress, freed of one source of confusion; and so will it come to be founded on the only sound basis of classification—pathology.

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Reviews.

PROGRESS IN RADIOLOGY.

A SECOND edition of the well-known work "Recent Advances in Radiology", by Peter Kerley, is to hand.[1] This is really an entirely new book, as most of the information it contains has been rewritten and added to when advance is to be recorded. The book is arranged in most orderly fashion and is quite readable in spite of the condensation necessary to cover the large subject of diagnostic radiology in so small a book. The chapters on disease of the bones and joints are of particular interest, and in them will be found all the new work on advances in this class of work.

Ventriculography and encephalography receive attention, and the author points out that these procedures are by no means free from risk, and should be carried out only after all other diagnostic methods have been thoroughly explored. The indications for ventriculography are given as: (i) suspected brain tumour which cannot be diagnosed otherwise, (ii) hydrocephalus of obscure origin, and (iii) occasionally epilepsy. Correct technique and posturing are essential for good results, and a good knowledge of the normal is necessary.

Kymography in cardiac disease is likely to prove of value in the study of heart movements, especially in the distinction between transmitted and expansile pulsation; it is still in its infancy, however, and is far from perfect. Cardiac cinematography has not yet proved of great value. The author again warns workers that cardiac examination by X rays is only supplementary to a full clinical examination. The radiologist should never give a prognosis in cases of heart disease.

The chapters on pulmonary disease are very interesting and contain much new information. The author points out that in silicosis the only substance which produces the typical nodular fibrosis, when inhaled, is silica. There has been a definite increase in the number of cases of pulmonary carcinoma (which may occur in any part of the lung), while pulmonary sarcoma is extremely rare.

Gastro-intestinal disease is dealt with in a comprehensive manner, and the information given in a small chapter is quite equal to that found in an ordinary text-book. In small intestine obstruction valuable information may be obtained by noticing the presence of fluid levels in the distended coils of gut, and an opaque meal is often unnecessary. The magnesium sulphate method of examining the appendix is recommended, and by this method it is claimed that 100% of appendices may be demonstrated.

Chapters on cholecystography and urography are also of interest. A feature of the book is the excellent system of references to original papers on the different subjects. This book should be in the hands of all radiologists, and it should be of particular help to the country practitioner who has to do a certain amount of X ray work. The illustrations are of a very high class and should prove most helpful.

ORTHOPÆDIC SURGERY.

To the surgeon practising orthopædics, the general surgeon and the student, Dr. Mercer's book on orthopædic surgery will be equally welcome.[2] It is written by one who from wide experience in general surgery has turned to the narrower field of orthopædics, bringing with him the balanced judgement that only work in the larger sphere can produce. The book is not too large, and yet the descriptions of various conditions contain all the information which the reader can desire. Particular care has been taken to give classical procedures as well as the most recent work and the author's own views. The book is well produced, the illustrations, which are numerous, being beautifully clear and the numerous X ray reproductions excellent. It is a book for reference, but will certainly not be used for reference only. Dr. Mercer's descriptive style is so easy to read and his points are made so clearly that the difficulty is to put the volume down. There are only 906 pages, including a complete bibliography and index. Osteomyelitis, for example, occupies twenty pages, and yet this small space is sufficient to include a description of the bacteriology, source of infection, route, incidence of disease, pathology, course and history, clinical features, differential diagnosis and prognosis. Treatment of the various stages is dealt with, and there is a description of the following methods of after-treatment: Carrell-Dakin, Winnett-Orr, Albee's (bacteriophage), Baer's maggots, Stewart (picric acid and calcium carbonate), subperiosteal resection, treatment of bone cavities, planification, bone-filling operation, and the grafting method of Quick, of Melbourne. Several of these methods are quite recent, and Quick described his method of grafting osteomyelitic cavities only last year, so that one may judge how up to date is the information given. The author does not enthuse over maggots, as apparently the British surgeon, like the Australian, finds the method rather strong for his palate, however keenly the Americans speak of their results. Nevertheless the method is described and the theories underlying the success claimed for it are given clearly. The Winnett-Orr method, which many British surgeons find too disgusting for adoption, has been used successfully and enthusiastically by the author, as by most Australian surgeons. Although he describes it, he does not appear to have had personal experience of the use of bacteriophage in this connexion.

Set orthopædic operations for various conditions are described briefly yet clearly enough to give guidance to the surgeon whose personal experience may be insufficient to allow him to rely on in his choice of procedure. Any modification that the author has found useful is given.

What may be considered the essentially orthopædic conditions, such as congenital dislocations of the hip, deformities of feet, kyphosis and scoliosis, and the tuberculous affections of bones and joints are thoroughly dealt with, as are also many descriptions of apparatus and plaster technique. Only a few fractures are dealt with, space being unavailable for any systematic treatment of this subject.

From the general excellence of this volume and the information given by Professor Fraser in the foreword, that Dr. Mercer is particularly interested and has great experience in fracture work, it is to be hoped that one day he may write a book on their management, for in no branch of surgery is mature judgement such as his so valuable.

[1] "Recent Advances in Radiology", by P. Kerley, M.D., B.Ch. (N.U.I.), D.M.R.E.; Second Edition; 1936. London: J. and A. Churchill Limited. Demy 8vo, pp. 332, with 176 illustrations. Price: 15s. net.

[2] "Orthopædic Surgery", by W. Mercer, M.B., Ch.B., F.R.C.S., F.R.S., with a foreword by J. Fraser, M.D., Ch.M., F.R.C.S.E.; Second Edition; 1936. London: Edward Arnold and Company. Royal 8vo, pp. 917, with illustrations. Price: 40s. net.

Notes on Books, Current Journals and New Appliances.

FAVOURITE PRESCRIPTIONS.

A SERIES of eighteen articles on hospital pharmacopœias, originally published in *The Practitioner*, comprise a volume entitled "Favourite Prescriptions"[1] edited by Sir Humphry Rolleston and A. A. Moncrieff. Compiled chiefly for the guidance of the general practitioner, the book contains a wealth of prescriptions whose value has been tested by long experience. Moreover, historical interest attaches to many of them, some being associated with the names of famous physicians. The pharmacopœias described include those of famous hospitals, such as Saint Bartholomew's, Guy's, Saint Thomas's, The London, The Middlesex, Saint George's, the Dublin Hospitals, The Royal Infirmary of Edinburgh, and so on.

[1] "The Practitioner Handbooks: Favourite Prescriptions", edited by Sir H. Rolleston, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and A. A. Moncrieff, M.D., F.R.C.P.; 1936. London: Eyre and Spottiswoode Limited (for The Practitioner). Demy 8vo, pp. 227. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 27, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

AN AUSTRALASIAN COLLEGE OF PHYSICIANS.

In February, 1931, there appeared in this journal a short account of the first annual general meeting of the Association of Physicians of Australasia, held in Sydney on November 30, 1930. This was the first official announcement of the existence of this body. Commenting in a subsequent issue on the establishment of the association, we expressed the hope that from it a College of Physicians of Australasia would some day be evolved. That hope is shortly to be realized.

During the last six years the Association of Physicians of Australasia has flourished. Those who do not enjoy the privilege of membership have heard little of its doings, for it is a private society with a small and restricted membership; no representative of the Press, medical or lay, is admitted to its gatherings, and no report is furnished to medical journal or newspaper. Its members have striven to attain its objects—"the advancement of internal medicine and the promotion of friendship among physicians". If we can judge from verbal reports and from the obvious enthusiasm with which members travel from far and near to attend meetings, they have been successful. The forthcoming development is the logical outcome of the success that has followed the efforts of the last six years. At the inception of the association membership was limited to seventy-five; and members comprised those who were practising solely as physicians, particularly physicians attached to the large teaching hospitals, as well as others engaged in scientific work in one of the ancillary branches, such as pathology. The first president was the late Richard Stawell. Sir Charles Bickerton Blackburn succeeded Richard Stawell; and he was succeeded by Dr. S. V. Sewell, who at present holds the office. During the last year or two it has been realized that the fixed membership of seventy-five was inadequate and that expansion was inevitable. At a meeting of the association in Adelaide in September, 1934, the council of the association considered the matter and appointed a subcommittee consisting of Dr. S. V. Sewell, Dr. S. O. Cowen and Dr. L. S. Latham, to report upon the question of the formation of a College of Physicians for Australasia. At a general meeting held in April, 1936, it was agreed that the time was ripe for the foundation of a college and that a draft constitution should be drawn up. This work was carried out by Dr. S. V. Sewell, Dr. S. O. Cowen and Dr. J. G. Hayden, who received much assistance and valuable advice from officials of the Royal Australasian College of Surgeons. The draft constitution was approved by the association in October, 1936. While these preliminaries were being carried out, it was decided to extend the membership of the association, so that when the Association of Physicians takes the final step of constituting itself a college, it will have a membership of about one hundred and twenty-five. It is expected that the college will be constituted in a few months' time when the association meets in Melbourne. According to the draft constitution, the college will consist of fellows and members. So far no information is available of the qualifications necessary to secure election as a member; it has, however, been decided that fellows shall be elected from amongst the members. Provision has been made for the election of honorary fellows.

Readers will have learned long ago from newspaper reports that the building for the new college is to be erected in Sydney. Unfortunately some of the newspapers treated the matter as if it were one of interstate jealousy. We are glad to be able to state that the erection of the headquarters building in Sydney was the unanimous decision of the Council of the association when it met this month in Melbourne. The New South Wales Government has promised to give the sum of £25,000 towards the erection of a building, and the University of Sydney has made available an excellent site close to the old medical school. The Association of Physicians is to be commended for the deliberate fashion in which it has approached the creation of a college; there has been no undue haste, every move has been most carefully considered, and the six years' existence of the Association of Physicians has been in a sense an apprenticeship period for the college. The Council of the Australasian College of Physicians (to give it its correct title) should be able to avoid making the mistakes that are often associated with new ventures. It will have six years' experience of a voluntary association behind it, and it will surely be able to learn from mistakes that have been made by other bodies. If the new college has at heart the welfare of the whole medical profession in Australia as well as its own, it will take care not to clash in any way with the activities of the Branches of the British Medical Association, but rather to foster the closest possible relationship between the two bodies. It will recognize that no part of the profession, even the "senior service", can stand alone, and that its strength, as well as the strength of every other organization within the profession, depends chiefly on an active and flourishing British Medical Association.

Current Comment.

THE USE OF CHOLINE COMPOUNDS IN THE TREATMENT OF CHRONIC ARTHRITIS.

CONSIDERABLE attention has been paid of recent years to the vascular conditions accompanying chronic rheumatic infections. One of the most striking phenomena in chronic arthritis of the so-called rheumatoid type is the atrophic condition of the skin, depending in its turn upon local alterations in blood supply. Operations on the sympathetic nervous system have been carried out in numbers of these cases and good results are reported by some observers. It is obvious on reflection that one of the ends to be attained by any physical form of therapy is the improvement of the blood supply, and the same may be said of general measures, such as thermal therapy, even including the use of intravenous foreign protein. One of the most recent attempts in this direction is to utilize the known vaso-dilator properties of the choline derivatives.

D. Boyd, S. L. Osborne and D. E. Markson have reported their observations on the use of acetyl β-methylcholine chloride in chronic arthritis.[1] This compound is a synthesized product which is free from the nicotine-like action of acetylcholine; it is many times more active and can be given by mouth. These authors, however, have not been impressed by the results of oral administration and have also found that the systemic effects of the drug on the circulation may be unpleasant. Accordingly they have used it in a series of cases by the ionization method, using a 1% solution of the drug to saturate the active electrode. In this way they claim that the drug is introduced through the skin over the affected area which it is desired to treat. Evidence of this is forthcoming in the observation that the blood pressure characteristically falls and the pulse rate rises during the treatment, while the patient is also conscious of flushing of the face and of perspiration, even when the skin is habitually dry. These authors hold that the advantages of this method are the slight degree of general reaction produced and also the enhanced local effect. Plethysmographic records appear to show that in most cases the local blood supply is increased after the ionization; this has been observed, for instance, in the fingers. Twenty-seven patients have been treated over the period of two and a half years, and encouraging results have been obtained. As might be expected, the patients with the more advanced changes were less benefited, and those reacting most favourably were those with cold, moist and often cyanotic extremities. The authors are careful to disclaim any belief that there is of necessity a direct connexion between the circulatory changes and the arthritis, and they further admit that the well-known cycles of changes occurring in arthritics and the difficulty of any exact measurement of improvement make an accurate opinion as to the value of the treatment most difficult to express. Still, they have observed numbers of cases of improvement to the point at which functional capacity was at least partly restored to damaged joints.

This is, of course, only one more of the many methods used in the treatment of this most dis-

[1] *Annals of Internal Medicine*, December, 1936.

abling disease, but it is of value in pointing out to the medical profession the extremely important part played by physical therapy. It cannot be denied that sufficient interest in physical medicine has not been taken by the profession in past years, with the result that the little-trained and untrained irregular practitioners have stolen a march on the regular forces.

PERNICIOUS ANÆMIA.

BEFORE Minot and Murphy made their epochal discoveries, pernicious anæmia killed its victims as a rule within three or four years. In a few recorded cases the patients lived for seven or eight years, and in a few complete recovery was reported. In Cabot's series of 1,200 cases, ten had lasted seven years or more, and in six recovery had occurred; but it is reasonable to doubt whether any patient recovered from pernicious anæmia before the advent of liver therapy. There may have been mistakes in diagnosis. Such mistakes can occur quite readily. Macrocytic hyperchromic anæmia is not always true pernicious anæmia. Lesions of the spinal cord in pernicious anæmia have always been a source of interest and speculation. In Cabot's large series the cord was found to be affected in 84% of cases in which *post mortem* examination was performed. During the past few years many investigators have expressed their views concerning the possible prevention of cord lesions by liver therapy. There has been some diversity of opinion. This may be explained in several ways. For example, on the one hand, patients may not have been kept long enough or closely enough under observation; and, on the other hand, lesions may have been overlooked before the onset of treatment, and lesions due to syphilis or other cause, appearing during treatment, may have been mistaken for subacute combined degeneration. The cord lesions associated with pernicious anæmia presumably are caused by the same conditions that cause the anæmia; it would seem, therefore, that it should be possible to prevent their appearance by the same therapeutic measure that reestablishes normal erythropoiesis. This may be so; but its proof is difficult. There are many things apart from cause and effect to be considered. In some cases characteristic nervous symptoms appear before the anæmia, and it is possible that in many or most cases lesions of the spinal cord occur early in the course of the disease, without causing sufficiently pronounced symptoms to make their presence obvious.

William Needles has made a study of liver therapy in the prevention of subacute combined degeneration of the spinal cord.[1] Of 30 patients with pernicious anæmia he had to eliminate 17 from his consideration, because of the existence of some other possible cause of organic nervous disease, or because of the patients' neglect of adequate liver therapy, or because some evidence of subacute combined degeneration existed before the commencement of treatment. He points out that the evaluation of liver therapy cannot be made unless, at the commencement of treatment, patients are free of organic nervous disease and of any conditions that may cause abnormal neurological signs, and unless they take liver in adequate doses, as determined by the erythropoietic response. A difficulty in forming an opinion as to the value of treatment is that old people (those most frequently affected with pernicious anæmia) are apt to suffer from nervous lesions, perhaps due to vascular disease, that may be confused with subacute combined degeneration. In four of Needles's cases there was no evidence of involvement of the nervous system after periods ranging from four and a half to eight and half years after the onset of symptoms. In six cases there was very slight evidence of nervous damage after periods ranging from four to nine years after the onset. In two cases slight nervous damage, present at the commencement of treatment, did not alter to any great extent over the years of treatment, although in one case the duration of the disease was eight and a half and in the other six and a half years. In one case only was there evidence of severe nervous damage (six years after the onset of symptoms); but it was found that the patient had taken liver in inadequate doses. As we know what Needles means, we shall allow the following statement to stand; it is worth quoting: "The average duration of the illness for the patients who are still alive and well is six and one-half years." Needles points out that this greatly exceeds not only the time usually required for extensive involvement of the spinal cord before the advent of liver therapy, but also the usual duration of the illness in those days. As a result of his investigations he inclines to optimism concerning the value of liver therapy in the prevention of lesions of the spinal cord.

The results of Needles's investigation are encouraging. It is unfortunate that he had such a small series of cases. The tendency among British authorities is to the view that liver therapy is of great value in the prevention of subacute combined degeneration. This is probably the correct view. At any rate, it is the duty of every medical practitioner first to spare no pains in the recognition of pernicious anæmia, and secondly to treat it thoroughly.

[1] *Archives of Internal Medicine*, November, 1936.

THE ADELAIDE CONGRESS.

THE attention of readers is directed to the application form for membership of the fifth session of the Australasian Medical Congress (British Medical Association), to be held at Adelaide from August 23 to 28, 1937, published on page viii of the advertisements in this issue. Important information regarding travelling facilities will be found on page 345. Intending members of congress are invited to complete the application form without delay.

Abstracts from Current Medical Literature.

MEDICINE.

Malaria.

J. A. SINTON (*The Indian Medical Gazette*, April, 1936) discusses what he terms man-made malaria. In India there are numerous instances where malaria has been introduced or its prevalence greatly increased by the activities of man. "The inhabitants of India do not appear to be content with the large amount of malarial sickness with which Nature has provided them, but, in many instances they would seem almost to have made special efforts to increase the disease." Harmful conditions may be due to the ignorance or neglect not only of private individuals and communities, but also of industrial concerns and even government departments. Factors increasing the prevalence of malaria are the following: (i) measures resulting in an increase in the numbers of anophelene mosquitoes; (ii) the introduction of human carriers; (iii) conditions leading to economic stress and a lowered resistance of the population. These factors are usually seen in combination. Measures resulting in an increase in the numbers of malaria-carrying mosquitoes may be local or widespread. Local activities are domestic or communal, and include: inadequate drainage; neglect of stagnant water; the choice of an improper site for a town; building operations; inadequately attended irrigation; over-irrigation; construction of docks, harbours, waterworks *et cetera* without proper sanitary precautions; indiscriminate opening up of jungle, *et cetera*. Remedies consist in education of the masses, legislation and the institution of anti-mosquito measures. The more widespread activities leading to increase in the prevalence of malaria are mainly concerned with engineering works. The author points out that, while many of these works have added to the prosperity of the inhabitants of certain areas, in other cases they have eventually had a deleterious effect on both the economic status of the people and their health. The potentialities of water for breeding large number of mosquitoes depend very often on the amount of water edge in relation to the volume of water; when a body of water is so large that the "edge in proportion to the exposed surface is infinitesimal, extensive breeding is . . . inhibited". While decrease in water (through drainage *et cetera*) may be of value in some circumstances, it may have dangerous effects in others. Activities that increase mosquito-breeding by increasing water are as follows: (i) excavations, (ii) obstruction to natural drainage, (iii) an increased influx of water without the provision of drainage, (iv) the introduction of large irrigation projects without the provision of a proper drainage system at the same time. As an example of increased mosquito-breeding as a result of decrease in water, the author mentions the conditions in Lower Bengal, where formerly large areas were inundated by water from the Ganges at certain seasons; with the construction of embankments this type of irrigation has been stopped, with the consequence that malaria has increased and the land has become impoverished. The author remarks that any large industrial, engineering or mining project in India usually necessitates the importation of labour. "These large 'aggregations of tropical labour' frequently suffer severely from malaria when introduced into a malarious area, or they may import malaria into a relatively healthy tract." In some instances it has been found impossible to complete the work without anti-malarial operations. The imported labourers may spread the disease to the surrounding inhabitants. Increased prevalence of malaria may continue after the completion of the work because of physiographical changes *et cetera*. In some parts of the country, as in Bengal, extensive engineering works have caused impoverishment of the soil, and consequently poverty and lowered resistance of the inhabitants. The relationship between these conditions and malaria has been stressed by various observers. The author states: "The widespread presence of man-made malaria is a very serious problem in India."

The Blood Iodine Level in Hyperthyreoidism.

STUDIES in the blood iodine level and the urinary excretion have demonstrated that in hyperthyreoidism there is a progressive depletion of the iodine content of the thyreoid gland concomitant with an increase in the iodine in the blood and urine. H. J. Perkin and L. M. Hurxthal (*New England Journal of Medicine*, October 15, 1936) observed that in a series of over one hundred cases of hyperthyreoidism with an elevated blood iodine there may be as much as 0·25 to 1·00 milligramme of iodine in the urine per day; the average diet contains considerably less than 0·25 milligramme of iodine, so it seems likely that a hyperthyreoid individual with an elevated blood iodine is in a state of negative iodine balance. The writers put forward the hypothesis that, should a hyperthyreoid syndrome persist in an individual, the thyreoid gland will eventually become depleted of its store of iodine, at which time the blood and urinary iodine return to normal. The present study was undertaken to determine the influence of subtotal thyreoidectomy on the blood iodine level and to ascertain, if possible, whether 'an elevated and a normal blood iodine were each a definite state of iodine metabolism in the hyperthyreoid individual. Accordingly the blood iodine level was followed post-operatively in 141 cases in which the pre-operative level had been determined. From the tabulated results it is seen that the average blood iodine level of hyperthyreoid persons with an elevated pre-operative level returned to normal within three months after operation, and throughout the time followed (twelve months) was found to remain normal; clinically there was no evidence of recurrent or persistent hyperthyreoidism in any of these cases. In the hyperthyreoid cases with a normal pre-operative blood iodine content, the average blood iodine value was increased three months after operation, reaching a peak of elevation at six months, then gradually diminishing to normal at the end of twelve months; a small proportion of these patients have shown signs clinically of persistent or recurrent hyperthyreoidism. The conclusion is drawn that subtotal thyreoidectomy in the early stages of hyperthyreoidism is inhibitive of recurrence of the syndrome. If the blood iodine is normal, a more radical subtotal thyreoidectomy may reduce the incidence of recurrent hyperthyreoidism. However, the possibility should be considered that, since the tissue removed from the latter type of case generally showed characteristic histopathological change, regeneration of such tissue left at operation may give rise to an increase in the blood iodine level, together with the manifestations of recurrent hyperthyreoidism. It is stated also that, on the basis of the results obtained, the blood iodine level is indicative of a condition of iodine metabolism and, when influenced by abnormal thyreoid function, is of interpretable value, both before and after subtotal thyreoidectomy.

The Leucocytic Formula as a Prognostic Guide in Pulmonary Tuberculosis.

PIERRE BOURGEOIS AND BOQUET-JESENSKY (*La Presse Médicale*, November 7, 1936) have made a study of the white cell counts of 200 sanatorium patients at regular intervals, as a possible guide to their progress. They recognize Medlar's subdivision into three characteristic types: (i) The septic, with a polynuclear leucocytosis, lymphocytes under 25% and monocytes above 8%; the ratio of lymphocytes to monocytes progressively diminishes as the condition of the patient deteriorates. (ii) The resistant type, in which leucocytes are about 10,000 per cubic millimetre, the lymphocytes tend to approximate the polynuclear cells in numbers, monocytes are below 10%, and the lymphocyte : monocyte ratio is constantly elevated. (iii) The hyperplastic type, with a normal number of leucocytes, lymphocytes 25% or more, and monocytes below 10%. In this group a monocytosis indicates fresh activity, and a lymphocytosis a tendency to healing. The authors point out that great variations may be found in the counts. They regard the lymphocyte: monocyte ratio as the most sensitive

index to the patient's progress, and as a better guide than either the Vernes test or the sedimentation rate.

Radial and Coronary Arterial Changes.

S. W. SAPPINGTON AND HUNTER S. COOK (*The American Journal of the Medical Sciences*, December, 1936) report observations on changes in the radial artery, both at various age periods and as a result of sclerosis, in comparison with similar changes in the coronary and other muscular arteries in the same case. The radial artery has received particular attention as an index of general or visceral arteriosclerosis, and most written opinions seem to favour the view that the clinical condition of the radial artery offers no accurate information as to arterial conditions elsewhere. The large coronary arteries show, both as to incidence and degree, among the greatest changes of any arteries in the body; this is especially the case with reference to muscular arteries, of which the coronary is an example. The coronary arteries from birth to death gradually thicken, almost exclusively as a result of intimal change. Up to forty years this is roughly uniform and may be considered hyperplastic and physiological. After forty there is not much change of this type, but commonly supplementary eccentric additions are made to the intimal thickness by atherosclerotic formation. This may be totally expressed in the altering ratio of intimal to medial thickness, and the altering ratio may be considered as an index of coronary sclerosis, physiological or pathological. Radial artery changes have been analysed along the same lines; the maximum change here, consisting of a roughly uniform intimal collar of elastic and fibrous connective tissue and achieved between forty-five and sixty-five years, is comparable in kind, but not in degree, with the probable physiological variety seen in the coronary artery before forty years. Certainly the striking extreme eccentric atheromatous thickening so characteristic of the coronary is not seen in the radial artery. These differences in radial and coronary vessels are strikingly brought out in a comparison of the ratios between the intima and the media in the two vessels, as the changes in both arteries are dominantly intimal. In the coronary, the intimal-medial ratio soon after birth is 1 to 6 or 8; in the third decade they are commonly 1 to 1, whereas in the sixth decade ratios of 5 or 10 to 1 are common. In the radial the ratio of 1 to 8 found in the first decade shifts much more slowly, until it is 1 to 4 or 5 in the fourth decade; later, the ratio may be 1 to 2 or 3, only occasionally 1 to 1. Mönckeberg's sclerosis is a disease of muscular arteries, particularly of the peripheral vessels, and is much more common in the lower extremity; the change is essentially one of medial calcification, and is seen in the muscular arteries, atherosclerosis attacking the elastic arteries. From the observations made, it was concluded that radial calcification was almost invariably medial; on the other hand, medial calcification of the coronary artery was scarcely seen, whereas intimal calcification was very common. Scrutinized as to the relation of certain diseases to arteriosclerotic change, it was found in the small series available that cases of diabetes and benign and malignant hypertension showed pronounced or extreme coronary sclerosis, while the corresponding radial artery was little affected. In six cases of clinical coronary disease little radial change was apparent. Advanced coronary sclerosis was also unexpectedly encountered in a number of cases in which the preceding history and disease seemed entirely unrelated to vascular change. In such cases coronary disease was not suspected during the life of the patient; certainly the radial artery gave no information.

Achlorhydria.

A. MOLTKE AND A. S. OHLSEN (*The Lancet*, October 31, 1936) describe investigations in chronic and subchronic proliferative arthritis. The Ewald test meal was given, fractional analysis of the gastric contents was carried out, the histamine test being used. Sixty-nine patients with true rheumatoid arthritis or proliferative arthritis were studied. There were 45 females and 24 males. True achlorhydria was noted in 23% of these patients. Of women between forty and fifty-nine years of age, 50% showed achlorhydria. This is greatly in excess of the normal percentage for women of that age. Generally, achlorhydria was more frequent among these patients than among normal people; this may have some ætiological significance.

Pituitary Function.

B. ZONDEK (*The Lancet*, October 10, 1936) describes experiments demonstrating impairment of anterior pituitary functions by follicular hormone. Œstrin in small doses stimulates, while large doses depress the function of the anterior pituitary. Further, injection of 100,000 mouse units of œstrin in a normal woman, between the sixth and twenty-fourth day of the menstrual cycle, will prevent the onset of the next period, by its action through the anterior lobe of the pituitary. Young rats were given 5,000 mouse units of "Dimenformon" twice a week; at the end of three or four months these rats were stunted in growth. This stunting of growth could be abolished by administration of growth hormone. Similar effects were produced by applying "Dimenformon" to the skin of rats. In chickens, 50,000 mouse units of "Dimenformon", injected twice a week, caused a retardation of growth and lack of development of the comb in cocks, the testes atrophied and spermatogenesis was absent. In rats again, the scrotal sac disappeared and the penis and testes remained infantile. In females the ovaries were very minute and contained no *corpora lutea*. Considerable changes in bone structure were also observed, epiphyseal junctions remained open, the spongiosa was filled with porous tissue, only negligible remnants of the medullary cavity being left. When œstrin treatment was discontinued after several months, the animals did not spontaneously resume their growth. However, if Evans's pituitary growth hormone was administered, they began to grow and gain weight. This showed that the dwarfing was of pituitary origin. These experiments show that œstrin affects the anterior pituitary, diminishing its functions, and especially the gonadotropic and growth hormones.

"Thorotrast."

F. R. SELBIE (*The Lancet*, October 10, 1936) describes the experimental production of sarcoma with "Thorotrast". This substance, a colloidal solution of thorium dioxide, is extensively used in radiology for demonstration of blood vessels, cerebral ventricles, liver and spleen. Subcutaneous inoculations of 0·3 cubic centimetre of "Thorotrast" were made in sixty rats, on two occasions at fortnightly intervals; sixty mice were inoculated with 0·1 cubic centimetre at the same time. During fourteen and a half months, fourteen tumours had appeared in the rats, all spindle-celled sarcomata. In the mice only two tumours had appeared. The growths were attributed to radioactivity of thorium dioxide and its disintegration products. Mesothorium and radiothorium, two of the main disintegration products of thorium dioxide, are two of the main constituents of the luminous paints, which by ingestion produce fatal poisonings and osteogenic sarcomata in dial painters. In view of the danger of its causing malignant disease, "Thorotrast" should never be introduced into the human body.

Crystalline Insulin.

S. S. ALTSHULER AND R. LEISER (*The Journal of the American Medical Association*, November 14, 1936) describe results of using a new crystalline insulin. Standard insulin and crystalline insulin were injected in doses of 10 units into 10 normal non-diabetic subjects. The blood sugar was noted before and at intervals after the injection. It was observed that the crystalline insulin lowered the blood sugar more gradually and maintained the low level for a longer period than the standard insulin; at the end of seven hours the blood sugar was still below fasting level. Twenty-nine diabetic patients were tested with standard and crystalline insulin. Comparative studies showed that the crystalline insulin controlled the diabetes with fewer units given in fewer doses. The effects of crystalline insulin lasted for thirteen or fourteen hours in diabetics.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Lewisham Hospital, Sydney, on August 20, 1936. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Neuro-Surgical Cases.

DR. N. W. G. MACINTOSH, on behalf of DR. GILBERT PHILLIPS, demonstrated twelve neuro-surgical cases. The cases were exhibited in three groups, namely: operation cases, cases of severe cerebral trauma, and cases for diagnosis.

The first patient had been referred in May, 1934, by Dr. Conrad Blakemore, and his condition was diagnosed as a mixed adenoma of the pituitary gland. (This case was reported in THE MEDICAL JOURNAL OF AUSTRALIA of November 24, 1934, at page 686). The patient afforded an example of the value of transsphenoidal pituitary decompression in a slowly growing tumour of this nature. During the two years since operation the patient had been quite well and had been carrying on his usual occupation as a salesman.

The next patient had an unverified cerebellar tumour. The patient had been referred by Dr. John O'Brien, and a suboccipital decompression had been performed twelve months previous to the meeting. In view of the patient's condition and the very high intracranial tension, no search was made for the neoplasm at operation. After operation, papilœdema and diplopia disappeared, and the patient, who had previously been so ataxic that he was unable to walk, returned to school and had put on 15·75 kilograms (two and a half stone) in weight during the twelve months before the meeting. The interesting contrast between the skiagrams of the *sella turcica* in this case and in the previous one was pointed out. In the former, the *sella turcica* was enlarged and the *dorsum sellæ* and the posterior clinoid processes were almost completely eroded. In the latter, the classical picture produced as the result of increased intracranial tension was seen, namely, "ballooning" of the *sella turcica* without erosion of the *dorsum sellæ*.

The next patient, who had a perineural fibroblastoma of the eighth cranial nerve, had been referred by Dr. R. J. Murphy. A complete intracapsular removal of the tumour had been performed, and the interest of the case lay in the fact that complete infranuclear facial weakness had appeared in a single night and auditory symptoms had been present for a few months only. (This case was reported in detail in the issue of November 14, 1936, of THE MEDICAL JOURNAL OF AUSTRALIA.)

Dr. Macintosh next showed two patients who had suffered compound comminuted depressed fractures of the skull. These patients were contrasted by the fact that operation had been performed immediately on admission in the first case and was delayed for some weeks in the second. It was pointed out that the two main factors determining the urgency of operation in compound depressed fracture of the cranial vault were the state of the wound and the likelihood of penetration of the *dura mater*. It was known that post-traumatic sequelæ were about ten times more common after rupture of the *dura mater*, and it was believed, therefore, that, unless the overlying wound was grossly contaminated or the *dura mater* was obviously or probably penetrated, operation for the elevation of the depressed bone fragments might be delayed until the patient had recovered from the effects of any coincident cerebral damage. In the case in which operation had been delayed, the wound was clean and the skiagram of the skull showed the presence of a "pond" type of depression. This patient, as well as the other who had suffered a skull fracture with a grossly contaminated scalp wound as the result of falling off an electric train travelling at thirty miles per hour, was quite well, had returned to his usual occupation, and had experienced no post-traumatic sequelæ in the year succeeding operation.

Two patients who had had acute cerebral trauma (these cases were reported in detail in THE MEDICAL JOURNAL OF AUSTRALIA of May 23, 1936) were shown with the object of demonstrating the absence of relationship between unconsciousness and increased intracranial tension. In one case stupor had been prolonged for eleven days with only a slight increase in intracranial tension; in the second case unconsciousness was transient, but a considerable increase in intracranial tension (250 millimetres of cerebro-spinal fluid) persisted after the patient became conscious.

A series of five patients, presenting either frontal, temporal, parietal or occipital lobe symptoms or signs, were then exhibited for diagnosis.

Superior Thoracic Ganglionectomy.

DR. N. D. ROYLE demonstrated the effects of superior thoracic ganglionectomy on various conditions.

His first case was one of disseminated sclerosis. The patient, a male aged twenty-eight years, first complained of want of control of movements. He had tremor of the upper limbs and swayed when he walked. He had frequency of micturition and his speech was slurred. He had no balance in either lower limb and gave a double Babinski reaction. He also had loss of power on the left side.

After the performance of right superior thoracic ganglionectomy his speech became clearer. Frequency of micturition disappeared and his balance improved so that he could stand on his left lower extremity. The tremor also disappeared from his right extremity, and his left lower extremity became stronger than his right. Similar results on the right side followed the operation of superior thoracic ganglionectomy on the left side. His eyes, which were proptosed before the operations, assumed their normal position. His knee jerk, which was abnormal in postural response, also became normal. The patient stated to the assembled medical men that he no longer suffered from vertigo, which had affected him previous to the operations.

Dr. Royle's second case was one of spastic paraplegia. The patient was a male, aged eight years. When first seen, at the age of four and a half years, he did not sit up and could not stand without support. He had adductor spasm when he attempted to walk and had no balance in either of the lower extremities. His knee jerk was exaggerated in phasic response and the postural response was prolonged.

After the performance of superior thoracic ganglionectomy in 1931 the patient walked unaided. Dr. Royle explained that the effects were contralateral, that is, a superior thoracic ganglionectomy performed on the left side reduced spasticity on the right side and *vice versa*. That was, in his opinion, due to the effect of the operation on the vasomotor nucleus which controlled the vasomotor centres of the opposite side of the spinal cord. The depression of function of the vasomotor nucleus led to lessened constriction of the vessels of the spinal cord and so increased capillary flow.

Dr. Royle's third case was one of chronic hydrocephalus. The patient was a male, aged eighteen years. His head measured 67·6 centimetres in circumference and, according to his parents, was increasing in size. He had previously suffered from spastic paraplegia, for which lumbar ramisections on both sides had been performed. This improved his balance so that he could walk, but he still had a tendency to drag his right foot.

The operation of superior thoracic ganglionectomy was performed in 1932. Frequency of micturition, which had previously affected the patient, disappeared. The measurements taken of his head with calipers and steel tape showed a reduction in circumference of 0·6 centimetre in eighteen months. Dr. Royle expressed the opinion that the reduction in circumference was due to relief of œdema of the soft tissues, as the bone could hardly shrink in a patient of this age.

Fracture-Dislocation of the Neck of the Radius.

DR. R. V. GRAHAM showed a male patient, aged twelve years, who fell on his left elbow on July 16, 1936, and sustained a fracture of the neck of the radius, the head being displaced forwards and laterally so that the upper fragment was lying at right angles to the shaft. As an attempt at closed reduction failed, the radio-humeral joint was opened and the upper fragment levered back into place. As reduction was accomplished there was a definite click, and it was then found possible to get full flexion and extension of the elbow joint without any displacement of the fragment. The wound was closed in layers and the elbow supported in flexion and supination by means of strapping. Pronation and supination were permitted in the early stages and gradual extension of the elbow joint after one week. There were now good clinical union and practically full movement at the elbow and radio-ulnar joint except for a slight limitation of supination.

Compound Fracture of Tibia and Fibula.

Dr. Graham's second patient had been admitted to hospital on August 2, 1936, suffering from a compound fracture of the upper third of the tibia and fibula. The upper end of the lower fragment had penetrated the skin on the antero-medial aspect of the leg, and about one and a half inches of tibia protruded. On investigation of the deep tissues it was found that all the muscle attachments to the upper third of the distal fragment had been separated. The skin edges were excised, tags of fibrous tissue snipped off, and an aqueous solution of acriflavine was swabbed into every crevice, including the fractured ends of the bones. The fracture was then reduced by means of levers inserted between the fragments. The deep tissues were approximated and the wound was closed after ensuring that an excess of the acriflavine solution remained in the depths of the wound. The knee was encased in plaster from the heads of the metatarsal bones to the middle of the thigh. The temperature chart showed a low grade pyrexia, the temperature rising to about 37·8° C. (100° F.) in the evening for a few days only. A window cut in the plaster for purposes of demonstration showed complete absence of inflammation in the tissues, and it was shown that the limb could be moved in plaster with complete comfort to the patient. Skiagrams taken subsequently showed complete reduction of the fracture with the bones in good alignment.

Fracture of the Femur with Compound Dislocation of the Knee Joint.

Dr. Graham's next patient had been admitted to hospital in February, 1936, having been involved in a collision between a motor cycle and car. There was a comminuted fracture involving the upper third of the femur with medial displacement of the middle fragment and a compound dislocation of the tibia on the same side backwards, the wound running transversely across the popliteal space for about four inches. This patient also had a fracture of the sacrum and of the ulna. On investigation of the wound behind the left knee joint it was found that the knee joint was completely disorganized, the capsule being torn from its attachment to the femur, the crucial ligaments being torn and the heads of the gastrocnemius muscle being completely separated from the condyles of the femur. The fracture of the femur was reduced by manipulation, a Kirschner wire being inserted through the condyles for subsequent traction. The tissues in the popliteal space were swabbed over with acriflavine solution and the knee joint was filled up with it. It was found that the popliteal vessels and nerves were stretched across the back of the femur and they appeared to be the only tissues in the popliteal space which had not been lacerated. The dislocation was reduced by extension in the line of the limb and the wound was closed, the limb being supported on a Thomas splint with the knee joint in semi-flexion, and thirty pounds of extension were applied to the femur through the Kirschner wire. Provision was made for limited movements of the knee joint during convalescence and the patient was allowed up on a walking caliper early in May, 1936. The caliper was discarded towards the end of July and there was now sound union in the femur, with excellent alignment and no shortening, the knee joint being capable of full extension and flexion to a right angle with practically no instability; the sole residual instability was a slight lack of locking in extension.

Cavernous Hæmangioma of the Leg.

Dr. Graham then showed a patient who had complained of a large soft mass involving the medial and posterior aspects of the lower third of the right leg; the mass had been present for thirteen years and largely disappeared when the patient reclined. The swelling was so great that the patient found it difficult to wear an ordinary shoe for any length of time, although on first arising the shoe could be worn. About the middle of June, 1936, eight or nine tracks had been burnt into the tumour from various points around its circumference in directions radiating towards the centre by an endothermy needle at sufficient heat to obliterate the blood vessels during its passage. The scar tissue developing round these tracks had resulted in such pronounced occlusion of blood vessels that the tumour was already reduced to less than a quarter of its original size and was still diminishing. Should the decrease in size become stationary, it was proposed to repeat the operation, but at present it did not appear that this would be necessary.

Rupture of Ligament of the Knee Joint.

Dr. Graham's last patient, on July 2, 1936, had been struck on the lateral aspect of the right thigh by a motor cycle and suffered immediate disablement, with pain on the medial side of the leg and loss of power to support his weight on the extended knee joint. There was pronounced tenderness over the attachment of the medial collateral ligament to the condyle of the femur and an increase in mobility of the knee joint in both planes. The limb was placed in extension and a plaster cast was applied from the upper end of the thigh to just above the ankle joint, in which the patient walked about for six weeks. An X ray picture taken through the plaster showed that the tibial spine had been avulsed, the fracture line extending in an antero-posterior direction through each condyle and the loose fragment carrying with it the attachment of the menisci and crucial ligaments. The displacement, however, had been reduced when the limb was put in extension. The plaster was removed after six weeks and the patient now had active flexion of the knee joint from the position of full extension to about 135° and active extension to its full range. There was no undue mobility in either the lateral or antero-posterior directions.

Intussusception in an Adult.

DR. JOHN KENNEDY showed a female patient, aged eighteen years, who was first seen by him at 9.30 p.m. She complained of vomiting of sudden onset and of pain in the upper part of the abdomen.

There was a history of four previous attacks. Two attacks had preceded an appendicectomy performed in the country three months before the time of the meeting. The fourth attack occurred three weeks after the appendicectomy. At the onset of this attack the patient had a normal temperature, the pulse rate was 72 per minute, and no abnormal physical signs were present.

A hypodermic injection of 0·016 gramme (one-quarter of a grain) of morphine and 0·00043 gramme (one one-hundred-and-fiftieth of a grain) of atropine was given and relieved the pain, but the vomiting persisted through the night.

The following day, at about the same time, the temperature had risen to 37·25° C. (99° F.) and the pulse rate was 72 per minute. There were still no abnormal signs on examination. Calomel was given, followed by a saline aperient, and three bowel actions resulted.

The following day the pains recurred, vomiting had lessened, the temperature was normal, but the pulse rate was increasing, being from 112 to 120 per minute, and tenderness over and around the appendicectomy scar

region had developed and increased, with a feeling of tumefaction to palpation. An enema was given without result.

From the onset the patient had fear of intestinal obstruction; she was firm in her statement of the similarity of this attack to the previous attacks. A provisional diagnosis of acute intestinal obstruction was made.

Under "open ether" anæsthesia a mid-line suprapubic incision was made. Free blood-stained fluid was present intraperitoneally, and a large tumour mass of intestine filled the lower half of the abdominal cavity. The caecum was sought for and all the large intestine was found to be collapsed. The collapsed small intestine was traced proximally and the tumour mass was shown to be a large intussusception of the small intestine. The intestine involved in the intussusception was greatly engorged and reduction was impossible.

The mass was resected and a lateral anastomosis performed. One and a half pints of saline solution and glucose were administered intravenously before the patient left the operation theatre. The patient made a good recovery.

MEDICO-POLITICAL.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on February 3, 1937, PROFESSOR R. MARSHALL ALLAN, the President, in the chair.

Financial Statements.

DR. C. H. MOLLISON, the Honorary Treasurer, presented the statement of receipts and payments of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria for the year ended December 31, 1936. The statements are published herewith.

Dr. Mollison said that the statement of receipts and expenditure for the year under review disclosed that expenditure had exceeded revenue by approximately £309. Credit balances carried forward from 1935 amounted to £130 3s. 1d., and the debit balances in both accounts at December 31, 1936, amounted to £179 1s. 6d. There had been no falling away of subscriptions; on the contrary, they had increased by £276. An increase of expenditure and a loss of £100 from the British Medical Agency therefore accounted for the adverse position. Items of expenditure which showed an increase over 1935 were salaries, £413. This increase was due to a full year's salary being paid to the Medical Secretary for 1936 as against eight months for 1935, and during 1936 certain increases had accrued to other members of the staff. An amount of £82 for rebates to subdivisions also appeared in 1936. This was absent from the 1935 accounts. Travelling expenses, too, showed an increase of £42, for which visits to country centres by the Medical Secretary on Branch business was responsible.

The position was one which the Finance Subcommittee had under consideration. As it was not likely that any appreciable saving could be made in expenditure, some additional source of revenue, it was felt, must be looked for to enable the Branch to function properly and to meet its commitments. The Council was now assuming new activities in the interest of members, and these activities unfortunately involved increased expenditure. The Council had been very active regarding the interests of its members in workers' compensation, national insurance and infectious diseases, and this had involved heavy postage, much stationery and a considerable sum in travelling expenses.

The Finance Subcommittee did not view the position with any great alarm, but rather regarded it as one that needed careful watching and action in the future to bring about an adjustment.

Reference to the statements would show that the Branch was again indebted to the British Medical Insurance Company. During the year the company provided £268

BRITISH MEDICAL ASSOCIATION (VICTORIAN BRANCH).

Statement of Receipts and Payments for Year ended December 31, 1936.

RECEIPTS.	£	s.	d.
To Balance at National Bank of Australasia Limited at January 1, 1936	9	12	8
„ Cash in hand at January 1, 1936	12	0	0
„ Medical Benevolent Fund	180	12	0
„ Typewriting	17	9	11
„ Lists of Members	1	1	8
„ Subscriptions	5,737	7	6
„ Rent	23	3	0
„ Hire of Lantern	3	3	0
„ University Appeal	152	5	0
„ Stawell Memorial Fund	49	8	0
„ Sundries	22	17	2
„ Balance at National Bank of Australasia Limited at December 31, 1936	122	0	10
	£6,331	0	9

EXPENDITURE.	£	s.	d.
By Advertising	7	9	3
„ Salaries	786	15	2
„ Audit Fees	5	5	0
„ Bank Charges	1	6	4
„ Medical Benevolent Fund	187	12	6
„ Federal Council	122	0	0
„ Medical Society of Victoria	2,672	7	7
„ Printing	12	12	8
„ Postages	98	6	2
„ Repairs and Replacements	5	2	0
„ Stationery	35	15	4
„ Lanternist	3	10	0
„ Sundry Expenses	51	17	9
„ Telephone and Telegrams	30	7	4
„ *The British Medical Journal*	1,707	19	0
„ C. S. Crouch—Pension	300	0	0
„ Commission	6	0	2
„ Rebates to Subdivisions	81	11	6
„ University Union Appeal	152	5	0
„ Stawell Memorial Fund	49	8	0
„ Car Attendant	1	10	0
„ Cash in hand	12	0	0
	£6,331	0	9

Compared with the Books and Accounts of the British Medical Association (Victorian Branch) and found to be in accordance therewith.

EDGAR H. WARD, Financial Secretary.
C. H. MOLLISON, Honorary Treasurer.

J. V. M. WOOD & COMPANY,
Chartered Accountants (Aust.).

Melbourne, January 19, 1937.

MEDICAL SOCIETY OF VICTORIA.

Statement of Receipts and Expenditure for Year ended December 31, 1936.

RECEIPTS.	£	s.	d.
To Balance at National Bank of Australasia Limited, January 1, 1936	120	10	5
„ Subscriptions	2,672	7	7
„ British Medical Insurance Company—Grant	480	0	0
„ Hospital Benefits Association—Repayment of Part Loan and Interest	176	11	10
„ Balance at National Bank of Australasia Limited at December 31, 1936	57	0	8
	£3,506	10	6

EXPENDITURE.	£	s.	d.
By THE MEDICAL JOURNAL OF AUSTRALIA	1,327	2	6
„ Rates, Taxes and Insurance	23	14	2
„ Repairs	24	14	11
„ Debenture Interest	302	10	0
„ Medical Secretary—Salary	843	15	0
„ Staff—Salaries and Wages	342	6	0
„ Audit Fee	5	5	0
„ Travelling Expenses	48	2	3
„ Bank Charges	1	10	0
„ Furniture and Equipment	1	7	6
„ Library—			
Books and Journals £429 8 2			
Less Purchases by B.M.I. Co. £268 0 7			
Purchases by Dermatological Assoc. 18 0 7			
286 1 2			
	143	7	0
„ British Medical Insurance Company—Repayment Part Loan and Interest	176	11	10
„ Postages	81	16	11
„ Stationery	35	15	4
„ Telephone	30	7	3
„ Electric Light	53	17	10
„ Printing	12	12	7
„ Legal Expenses	20	9	6
„ Entertainment	19	16	3
„ Commission	6	0	2
„ Sundries	5	8	6
	£3,506	10	6

Compared with the Books and Accounts of the Medical Society of Victoria and found to be in accordance therewith.

EDGAR H. WARD, Financial Secretary.
C. H. MOLLISON, Honorary Treasurer.
J. V. M. WOOD & COMPANY,
Chartered Accountants (Aust.).

Melbourne, January 19, 1937.

for the purchase of books for the library, and £480 as a direct grant towards the Medical Secretary's salary. Agency business unfortunately during the year had been poor and had made no contribution towards Branch revenue, but this was a source to which Dr. Mollison hoped they might confidently look in the future. The extent to which the Branch could benefit from the Agency Company and the Insurance Company depended upon the support these companies received from members. The success of the companies was a matter that should strongly appeal to members, because any advantage the companies gained would accrue to the Branch and so pass to members.

Dr. Mollison then formally submitted for adoption the audited statements of receipts and expenditure of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria. The statements were adopted.

THE KATHERINE BISHOP HARMAN PRIZE.

THE Council of the British Medical Association is prepared to consider an award of the Katherine Bishop Harman Prize in the year 1938. The value of the prize is £75.

The purpose of the prize, founded in 1926, is the encouragement of study and research directed to the diminution and avoidance of the risks to health and life that are apt to arise in pregnancy and child-bearing. It will be awarded for the best essay submitted in open competition, competitors being left free to select the work they wish to present, provided this falls within the scope of the prize. Any medical practitioner registered in the British Empire is eligible to compete.

Should the Council of the Association decide that no essay submitted is of sufficient merit, the prize will not be awarded in 1938, but will be offered again in the year next following this decision, and in this event the money value of the prize on the occasion in question shall be such proportion of the accumulated income as the Council shall determine. The decision of the Council will be final.

Each essay must be typewritten or printed in the English language, must be distinguished by a motto, and must be accompanied by a sealed envelope marked with the same motto and enclosing the candidates name and address.

Essays must be forwarded so as to reach the Medical Secretary, British Medical Association House, Tavistock Square, London, W.C.1, not later than December 31, 1937. Inquiries relative to the prize should be addressed to the Medical Secretary, Medical Department, British Medical Association House, Tavistock Square, London, W.C.1.

NOTICE.

THE following notice is published at the request of the Medical Secretary of the Victorian Branch of the British Medical Association.

Information has been received that some members of the Victorian Branch have been approached by insurance companies with the request that accounts for medical service to patients coming under the provisions of the *Workers' Compensation Act*, 1935, should be reduced and that the fee for visits after the first attendance should be 5s.

Members are particularly requested not to accept this proposal and to make no compromise with the insurers.

A further statement of the position under the Compensation Acts 1935 and 1936 will shortly be circulated.

Medical Societies.

PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE eleventh annual general meeting of the Public Medical Officers' Association of New South Wales was held at the British Medical Association House, 135, Macquarie Street, Sydney, on January 28, 1937.

Dr. E. L. Morgan occupied the chair, and twenty members were present. Seven forms of proxy were presented and accepted.

Annual Report.

The Secretary presented the annual report for the year 1936, which, after discussion, was adopted. The report is as follows.

1. The year 1936 has been more hopeful than recent years have been. The passing of the industrial depression is expected to be followed by progressive policies in all departments of Government with improved prospects for their medical staffs.

2. The Committee met eleven times during the year, the attendances of members being as follow: Dr. H. G. Wallace, 4; Dr. E. L. Morgan, 11; Dr. Madeline Gors, 8; Dr. W. K. Flook, 9; Dr. H. H. Nowland, 7; Dr. J. M. Rainbow, 9; Dr. H. V. Baret, 5; Dr. C. E. Percy, 11; Dr. J. Cooper Booth, 6; Dr. G. L. C. Saunders, 10; and Dr. H. H. Willis, 11.

Dr. Wallace, on being designated to act as Director-General of Public Health during the absence of Dr. E. Sydney Morris, resigned the position of President in April and Dr. E. L. Morgan was appointed to succeed him. Dr. J. Cooper Booth was elected by the New South Wales Health Department Group in place of Dr. Morgan.

3. The Association now has 100 members, of whom seven are retired members retained on the roll as honorary members. The death is recorded with regret of Dr. R. M. McMaster, Dr. J. S. Purdy and Dr. J. Reiach.

4. Three circular letters were distributed to members during the year.

5. It has been noted with pleasure that several of our retired members have been temporarily employed in various departments. It is felt that this practice might be extended with advantage to all parties. The practical goodwill of our members in this and other ways would do much to improve the lot of our retired colleagues.

6. As instructed at the last annual meeting, the Committee has made representations to the Medical Board of New South Wales about the Diploma of Psychological Medicine of the University of Sydney, with the result that that diploma is now registrable by the Board.

7. In April it was decided to cooperate with the Professional Officers' Association in its campaign for remission of the salary cuts under the financial emergency legislation, and in May a delegate was sent to a meeting of protest organized by the Teachers' Federation of New South Wales. Late in the year the New South Wales Government remitted portion of the cuts following complete remission in the Federal services in July.

8. The protest was continued against the new interpretation of Public Service Regulation 152 governing leave of medical officers in institutions under the New South Wales State Government. It is believed that there will be reversion to the practice of former years, but as a definite statement to that effect could not be obtained from any of the authorities concerned, continued vigilance will be necessary in the future.

9. The appeal for increased salaries by medical officers in mental hospitals was taken up by the Association and it was found necessary to brief counsel and to proceed in legal form. It is regretted that the attitude of the Public Service Board as at present constituted made this troublesome and expensive procedure necessary. The friendly negotiations of previous years would seem to be a much more satisfactory way of settling such disputes. After two hearings the Board made a determination giving slight increments to some senior officers, but none to juniors, and introducing several unfortunate anomalies. The Board has been informed that the Association considers the increments too few and too small to affect appreciably the unsatisfactory recruiting for this medical service.

10. It was found necessary to protest against the introduction of a system of censorship of the medical publications of medical officers in the Department of Mental Hospitals. It is considered that medical officers in the services should have full freedom to record their observations and publish articles in medical journals, the restrictions of the Public Service Regulations and Cabinet instructions being adequate to maintain departmental discipline.

11. It is regretted that an appointment has been made from outside a service which has blocked the promotion of some of our members. The Association has opposed such a practice in the past, and the Committee hopes to discuss the matter with the head of the department concerned in the new year.

12. The Committee decided that it had no objection to the system of honorary visiting specialists at State hospitals, attention having been drawn to the matter by a group of public medical officers in Victoria. It was also decided that this Association was uninterested in the propaganda on certain electrotherapists in New South Wales.

13. The Committee has been informed that the Repatriation Group of the Public Medical Officers' Association of Victoria contemplates forming a separate body registered as a union under the Federal *Arbitration Act*. The repercussions of this action on our Repatriation Group are being watched.

Financial Statement.

The Honorary Treasurer, Dr. C. E. Percy, presented the financial statement. It showed receipt of £38 0s. 8d. and disbursement of £30 5s. 4d., the chief item of which was £22 18s. 6d., being legal expenses of an appeal to the New South Wales Public Service Board. The statement was adopted.

Election of Office-Bearers.

The incoming committee was instructed to discover what alterations of the rules were necessary to permit of the election as president of a retired member of the services.

The following office-bearers were elected for the year 1937:

President: Dr. Oliver Latham.
Honorary Auditor: Dr. S. McGeorge.
Honorary Secretary: Dr. H. Hastings Willis.
Honorary Treasurer: Dr. C. E. Percy.

The following were elected by their respective groups as the committee for the year: Dr. M. Edelston-Pope and Dr. W. K. Flook (Education Department), Dr. G. L. C. Saunders (Works and Railways), Dr. H. H. Willis (Repatriation), Dr. H. H. Nowland and Dr. J. M. Rainbow (Mental Hospitals), Dr. J. Cooper Booth, Dr. J. McManamey and Dr. C. E. Percy (New South Wales Health Department).

Post-Graduate Work.

LECTURES IN SYDNEY BY PROFESSOR J. C. MEAKINS.

THE New South Wales Post-Graduate Committee in Medicine announces that a series of lectures and demonstrations will be given in Sydney during the general

revision course, May, 1937, by Professor J. C. Meakins, M.D., LL.D. (Edinburgh), F.R.C.P. (Canada), F.R.C.S., F.R.C.P. (London), Physician-in-Chief to the Royal Victoria Hospital, Montreal; Director of the Department of Medicine, Magill University and Magill University Clinic; late Christison Professor of Therapeutics, the University of Edinburgh. The programme is as follows:

Monday, May 24, 8 p.m.—The Great Hall of the University of Sydney: Formal admission of Professor Meakins to the Degree of Doctor of Medicine, *ad eundum gradum*; delivery of the second Sir Charles Clubbe Memorial Oration by Professor Meakins; presentation to Professor Meakins of the Sir Charles Clubbe Memorial Medallion by His Excellency Sir Philip Street, Lieutenant-Governor of New South Wales. Admission will be by invitation only. Any member attending the general revision course, or other medical practitioner, may obtain an invitation on application to the Secretary, the New South Wales Post-Graduate Committee in Medicine, the University of Sydney.

Tuesday, May 25, 3 p.m.—The Maitland Lecture Theatre, Sydney Hospital: "Circulatory Failure: Cause and Cure". A practical demonstration of cases exemplifying the various types of circulatory failure.

Wednesday, May 26, 8 p.m.—The Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street: Lecture entitled "Circulatory Collapse: Cause and Cure".

Thursday, May 27, 3 p.m.—The New Medical School, Medical Lecture Theatre: Practical demonstration of cases exemplifying the physiology and pathology of pulmonary failure.

Friday, May 28, 8 p.m.—The Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street: Lecture entitled "The Physiology and Pathology of Pulmonary Failure: Its Clinical Importance and Therapeutics".

Monday, May 31, 2.30 p.m.—New Medical School, Medical Lecture Theatre: Clinico-physiological demonstration (in association with Professor H. Whitridge Davies).

Tuesday, June 1, 8 p.m.—The Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street: Lecture entitled "The Systemic Significance of Renal Insufficiency".

Thursday, June 3, 8 p.m.—The Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street: Lecture entitled "Anæmia as a Common Disability".

Friday, June 4, 2.30 p.m.—Lecture Theatre, Royal Alexandra Hospital for Children: Clinico-pathological conference in association with Dr. F. Tidswell, Honorary Pathologist, the Royal Alexandra Hospital for Children.

Fees.

The fee for the eight lectures and demonstrations will be £3 3s., and for a single lecture or demonstration 10s. 6d. For hospital resident medical officers and those not engaged in active practice the fee will be £2 2s. For one week only (lectures and demonstrations) the fee will be £2 2s. For lectures only the fee will be £3 3s. All demonstrations will be part of the post-graduate afternoon course. The additional fee for the evening lectures to those attending the course will be £1 1s.

Congresses.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY.

THE fifteenth International Congress of Ophthalmology will be held at Cairo from December 8 to December 14, 1937. The two official subjects of the congress will be: (*a*) arterial hypertension of the retina, and (*b*) endocrinology and the eye. Papers will preferably refer to subjects related to these themes, and only a certain number of papers, of special originality and interest, may deal with independent subjects.

Any medical practitioner may become a member of the congress on payment of the fee of 50 Swiss francs. For associate members (relatives of members) the fee is 25 Swiss francs per person; but this does not entitle them to attend scientific meetings or to receive a report of the proceedings.

Two exhibitions have been arranged in connexion with the congress: (*a*) a scientific exhibition, comprising anatomical and pathological specimens or studies, photographs, X ray pictures *et cetera*; and (*b*) a commercial exhibition, comprising instruments, apparatus, drugs, printed matter *et cetera*.

Communications concerning papers for presentation at the scientific meetings should be sent to Dr. E. Marx, Oostzeedijk 316, Rotterdam, Holland. All other correspondence should be addressed to the Secretary-General, Dr. Mohamed Tewfik, XV Concilium Ophthalmologicum, P.O.B. Number 2001, Cairo, Egypt.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE inaugural meeting of the fifth session of the Australasian Medical Congress (British Medical Association) will be held on the evening of Monday, August 23, 1937. The congress will last until Saturday, August 28, 1937. Every member of the British Medical Association who intends to join the fifth session is requested to complete a form of application and to send it, together with two guineas, to the Honorary Local Secretary of Congress of the State in which he resides. Exchange must be added to cheques. It is important that application for membership of congress be made without delay, as there will be a great demand for accommodation and transport. Members are particularly requested to indicate whether they will be accompanied by ladies, and what type of hotel or boarding accommodation they require.

Travelling Facilities.

Railways.

Members travelling by any State or Commonwealth railways will be entitled to return tickets at single fares *plus* one-third. This concession applies to their wives, also sons under sixteen years of age and unmarried daughters.

The following are the fares from the respective capital cities:

	1st Class. £ s. d.	2nd Class. £ s. d.
Brisbane to Adelaide	13 0 0	8 13 4
Melbourne to Adelaide	4 13 4	3 2 8
Newcastle to Adelaide	9 3 4	6 2 8
Perth to Adelaide[1]	18 6 6	12 6 2
Sydney to Adelaide	8 6 8	5 10 8

Sleeping berth fees are as under:

	£ s. d.
Brisbane–Sydney	1 0 0
Sydney–Albury	1 0 0
Melbourne–Adelaide	1 0 0

(Pullman car, £1 5s.)

The reserved seat fees (either class) are as under:

	£ s. d.
Brisbane–Sydney	0 1 3
Sydney–Albury	0 1 0
Albury–Melbourne	0 1 3
Melbourne–Adelaide	0 1 3

Concession tickets issued to delegates are available for return within one month from date of issue.

[1] Including berths and meals, Western Australian and Commonwealth railways, and reserved seats, Port Augusta to Adelaide and return.

Shipping.

Members travelling by the Associated Steamship Owners' ships, that is, The Adelaide Steamship Company Limited, Melbourne Steamship Company Limited, McIlwraith McEacharn Limited, Huddart Parker Limited, Australasian United Steam Navigation Company Limited, and Howard Smith Limited, will be granted a concession of 10% off first class return fares. This also applies to their wives and adult members of families attending the congress.

The Tasmanian companies will also allow 10% off first class return fares for members and wives, but this does not apply to adult members of families.

The New Zealand shipping companies offer a 10% concession to delegates to congress accompanied by one female relative, providing ten or more travel from each port.

Airways.

Members desiring to proceed to congress by air will be allowed 10% off schedule fares, providing not less than ten members travel this way.

Correspondence.

"ENSOL."

SIR: In November, 1935, whilst working at some of the American clinics, I heard of the "Ensol" treatment for cancer. I therefore journeyed some hundreds of miles to make personal investigations.

The report on the effect of "Ensol" upon a transplantable mouse tumour by Moppett and Goldsworthy confirms my own findings; these were submitted to the South Australian Government by me as honorary Commissioner for Cancer Investigation in May, 1936.

No satisfactory evidence of "cures" was produced at the clinic, and my own findings were confirmed by J. B. Collip of Montreal, Max Cutler of Chicago, James Ewing of New York, and W. E. Gye of the Imperial Cancer Research Fund, London.

The corroborative evidence was from personal statements made to me by these gentlemen. Professor Gye (whom Dr. Connell was anxious for me to see regarding "Ensol") said that the method had been used on experimental animals in which cancerous implants had been made. There was no result whatever, and Professor Gye could see no rationale for the treatment.

It has been stated that this method has been used by practitioners in this country. This is unfortunate, as the curative qualities are quite inferior to others which are better known. Indeed it is doubtful whether this treatment has any place in the treatment of cancer at all.

Yours, etc.,

R. F. MATTERS,
M.D., M.S., F.R.C.S.E.

Adelaide,
January 16, 1937.

ARTIFICIAL PNEUMOTHORAX.

SIR: In your editorial of January 23 reference was made to the debatable question of the desirability of establishing thoracic surgical units for the particular surgical procedures adopted in connexion with pulmonary tuberculosis.

I would like to draw your attention to the fact that the Austin Hospital has in most respects assumed a position which is closely similar to that of special hospitals on the Continent. For the last five years the major portion of the operative work carried out on cases of pulmonary tuberculosis in Melbourne has been performed at the Austin Hospital, where a special unit for thoracic surgery has been developed, and has obtained special experience in the surgical procedures adopted in this condition.

While not depriving the acute general hospitals of a certain proportion of the work, the State Tuberculosis Bureau has adopted the policy of developing the Austin Hospital as a tuberculosis hospital, with facilities to carry out the operative work and after-care essential to cases in which surgical procedures on the thoracic cage are carried out. By this means the expense of establishing theatres and special staffs at sanatoria has been avoided, and an experienced thoracic surgical unit has been established in Victoria.

Yours, etc.,

A. F. CAMERON,
Manager and Secretary.

Austin Hospital for Cancer
and Chronic Diseases,
Heidelberg, N.22,
Victoria,
February 5, 1937.

HISTIDINE AND COAGULATION OF THE BLOOD.

SIR: In your issue of January 2, 1937, Dr. C. Stanton Hicks has reported the interesting fact that injection of histidine increases the rate of coagulation of blood 50% of its original value. Has Dr. Hicks considered the therapeutic use of histidine in hæmophilia and perhaps advancing cerebral hæmorrhage? I would be happy to hear his views on the subject.

Yours, etc.,

"PHYSICIAN."

Undated.

THE WASSERMANN TEST AND SYPHILIS.

SIR: In the journal of January 2 appears a statement by Dr. J. Morris Roe that a positive Bordet-Wassermann reaction—no matter how many times confirmed—has no absolute value and does not justify a diagnosis of syphilis.

The reasons given are, it seems to me, not very convincing, as a spirochætosis may include a huge variety of organisms, all of which surely will not give Bordet-Wassermann reactions similar to syphilis, even admitting a certain proportion of hypersensitive individuals.

Dr. Roe presumably uses the word "spirochætes" in the wide sense of the family Spirochætaceæ. Taking one recent classification, these may be divided into the genus *Spirochæta*, the genus *Cristospira* and the genus *Saprospira*.

The genus *Spirochæta* may again be divided into the *Spirochæta* (*sensu stricto*), the *Treponema*, and the *Leptospira*; and the *Treponema* may be further divided into the tissue parasites of syphilis and yaws and the blood parasites (*Borrelia*) of the relapsing fevers.

Now some of these spirochætes (Spirochætaceæ) are pathogenic in man, some in animals, some saprophytic, and others again free living; but Dr. Roe makes no distinction in so far that any of these may find their way into the "waters of rivers, lakes, marshes *et cetera* of tropical countries" and that people who drink these waters can become infected, developing a spirochætosis which may give a positive Bordet-Wassermann reaction.

Let us take just a brief list of the more common spirochætes with their habits and diseases, if any (spirochætes which, incidentally, can all be included in the more restricted genus *Spirochæta*).

Spirochæte.	Habitat.	Site.	Disease.
Treponema recurrentis ..	Man.	Blood.	Relapsing fever.
Treponema suis	Pig.	Blood.	Relapsing fever.
Treponema gallinarum ..	Fowls.	Blood.	Relapsing fever.
Treponema pallidum ..	Man.	Tissue.	Syphilis.
Treponema pertenue ..	Man.	Tissue.	Yaws.
Treponema cuniculi ..	Rabbit.	Tissue.	Rabbit syphilis.
Treponema schaudinni ..	Man.	Skin.	*Ulcus tropicum*.
Treponema vicenti ..	Man.	Throat.	Vincent's angina.
Treponema dentium ..	Man.	Mouth	(Saprophytes.)
Treponema mucosum ..	Man.	Mouth	
Treponema refringens ..	Man.	Genitals	(Saprophytes.)
Treponema gracilis ..	Man.	Genitals	
Leptospira icterohæmorrhagiæ	Man.,	—	Weil's disease.
Leptospira autumnalis ..	Man.	—	Akiyami fever type A.
Leptospira hebdomadis ..	Man.	—	Akiyami fever type B.
Leptospira biflexa ..	Water.	—	(Free living.)

Now how many of these will, if taken in man's drinking water, cause in him a spirochætosis with resultant positive Wassermann serology? Probably only the *Treponema pallidum* and *Treponema pertenuæ* to any marked degree, assuming they survive long enough to penetrate the alimentary mucosa.

Certain others, such as *Treponema recurrentis* and *Leptospira icterohæmorrhagiæ*, may cause mild degrees of positivity; but that any of the hosts of other spirochætes, pathogenic or saprophytic in birds or animals, or saprophytic in man, or free living, can cause a spirochætosis giving the usual positive Bordet-Wassermann serology of syphilis is a little far-fetched.

Spirochætes in some form or other are so ubiquitous and numerous that we must all harbour them in some form or other, and all of which probably can, as Dr. Roe states, be traced originally to a common ancestor. Yet the fact remains that probably less than 2% of the population would react positively to a Wassermann test properly performed.

Dr. Roe's statement "that the Bordet-Wassermann reaction has no absolute value" may be the result of his experience, and, if so, his observations and caution are to be commended, but he has in my opinion advanced a wrong explanation. Is it not far more likely that far too many serologists fail to realize that the positive Bordet-Wassermann reaction is a matter of degree rather than of specificity, and have failed to make use of sufficient refinement in technique and sufficient clinical controls?

The reaction may be divided into three groups:

1. Definitely negative.

2. Indeterminate—a serologic no-man's land where minor degrees of colloid instability may be due to numerous factors, such as leprosy, malaria, tuberculosis, Weil's disease, relapsing fever, and possibly other spirochætoses in the acute stages, for example Vincent's angina. These rarely attain a reaction titre sufficiently high to give definite positive reactions, but even when occasionally they do, the corresponding clinical picture of syphilis would give a positive titre immensely greater. A bad technique brings far too many bloods into this group, and further harm is done by many serologists employing an obsolete terminology. Instead of reporting such results as doubtful or indeterminate, they report them as positive (+1), positive (+2), positive (+3) *et cetera*. It is possibly "positive" Bordet-Wassermann reactions such as these to which Dr. Roe refers.

3. Definite positive reactions which on confirmation afford strong presumptive evidence of syphilis or yaws.

The point is that even with a careful technique the use of one or two known positive and negative sera as controls does not give sufficient demarcation between negativity, "no-man's land" or indeterminate reactions, and positivity. Unless a large number of known stages of positive and negative cases of syphilis are included as controls, the test loses much of its value, and such control is only possible where Wassermann tests are performed in close association and collaboration with a large venereal diseases clinic, as, for example, in the syphilis division of the medical clinic of the Johns Hopkins Hospital. This since 1914 was conducted as a separate entity, and the management of the Wassermann laboratory was placed in the hands of the director of the syphilis clinic. Any insidious trend towards over-obtuseness or over-acuteness in the serological tests must thus become immediately apparent, and so any reports sent out must have a very definite value and significance.

Stokes states that "the lack of these controls is responsible for the uncertainties into which many commercial laboratories and small private establishments have plunged the profession". With a thoroughly Wassermannized profession and an unduly large number of false positive reports from such laboratories, often further enhanced by obsolete and misleading terminology, the reason is at once apparent why cautious, observant physicians like Dr. Roe place little faith in a positive Wassermann as being indicative of syphilis, and why the syphilologist has often to decide between a Wassermannized physician, a syphilized patient, and an uncontrolled laboratory.

If and when all Wassermann tests are carried out with more adequate controls (such as is only possible in laboratories with access to much clinical material) then such cases will be fewer and the significance of the "positive" reaction will be more real, and Dr. Roe and many others in various centres will find that they will be able to place more faith in a positive Bordet-Wassermann reaction, which in high titre and on repetition affords almost incontrovertible evidence of syphilis (yaws excluded), and in lesser titre, provided certain obvious conditions are excluded, is strong presumptive evidence and is at least an indication for applying the therapeutic test of anti-syphilitic treatment.

An interesting and maybe important corollary of this discussion is the question of who is to bear the brunt in a case for damages for wrongful diagnosis.

If Dr. Roe's is the correct explanation, then the practitioner must accept the responsibility if the diagnosis is syphilis. On the other hand, if the above explanation is accepted, then it becomes a question of whether the serologist must take his share of the blame or whether the onus is still upon the practitioner to choose a reliable serologist.

Harrison, in the Medical Research Council's "System of Bacteriology" (1931) states definitely: "My own views coincide with those of Browning and Mackenzie as follows: with reservations which have little importance in temperate climates, a positive reaction is a specific sign of syphilis, and it indicates that the infection is, or has been, till very lately, alive in the patient's tissues. Similar views are expressed by Kolmer, who accepts yaws and possibly relapsing fever as liable to give positive reactions to a good method of the Wassermann test."

With the weight of such authorities behind him, it would seem that the practitioner might well be able to shift the blame and put the onus on the serologist to prove that his technique, if not the test itself, was above suspicion.

Yours, etc.,

Brisbane, January 11, 1937. G. S. HAYES.

PROFESSOR D. A. WELSH PRIZE FUND.

ADDITIONAL subscriptions have been received for the Professor D. A. Welsh Prize Fund as follows:

£5 5s.: Dr. J. E. Barling, Dr. B. T. Edye, Dr. W. M. Sinclair.
£3 3s.: Dr. J. Morton.
£2 2s.: Dr. E. M. Mackerras and Dr. M. J. Mackerras (jointly), Professor J. C. Windeyer.
£2 1s. 6d.: Dr. A. E. Colvin and Dr. C. S. Colvin (jointly).
£1 1s.: Dr. W. G. Armstrong, Dr. W. P. MacCallum, Dr. A. A. Palmer, Dr. H. Huff Johnston, Dr. C. W. W. Murray, Dr. Marie Hamilton, Professor J. B. Cleland, Dr. J. C. Storey, Dr. T. H. Small.

The total amount received now stands at £169 4s. 6d.

A RECEPTION IN LONDON FOR OVERSEAS MEDICAL VISITORS.

We are in receipt of a wireless message from the Royal Society of Medicine, 1, Wimpole Street, London, W.1, announcing the holding of a reception for overseas medical practitioners of the British Empire on May 10, 1937, at 8.30 o'clock p.m. Those desirous of attending should communicate with the Secretary of the Royal Society of Medicine before May 1.

WARNING NOTICE.

Members of the Branches are warned to peruse carefully and to understand fully the conditions of any contract form for the purchase of medical books *et cetera* before appending their signatures.

Books Received.

THE PRACTICE OF MEDICINE, by J. C. Meakins, M.D.; 1936. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay. Super royal 8vo, pp. 1357, with 505 illustrations, 35 of which are in colour. Price: 55s. net.

SYNOPSIS OF ANO-RECTAL DISEASES, by L. J. Hirschman, M.D., F.A.C.S.; 1937. St. Louis: The C. V. Mosby Company. Large crown 8vo, pp. 288, with 174 text illustrations and 6 colour plates. Price: $3.50 net.

TUBERCULOSIS, CANCER AND ZINC: AN HYPOTHESIS, by D. B. Cruickshank, L.R.C.P. and S., L.D.S., D.P.H., with an introduction by Sir P. Varrier-Jones, M.A., F.R.C.P.; 1936. London: Medical Publications Limited. Demy 8vo, pp. 86, with illustrations. Price: 7s. 6d. net.

Diary for the Month.

MAR. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 2.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 3.—Victorian Branch, B.M.A.: Branch.
MAR. 3.—Western Australian Branch, B.M.A.: Council.
MAR. 4.—South Australian Branch, B.M.A.: Council.
MAR. 5.—Queensland Branch, B.M.A.: Branch.
MAR. 9.—New South Wales Branch, B.M.A.: Ethics Committee.
MAR. 9.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 12.—Queensland Branch, B.M.A.: Council.
MAR. 16.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 17.—Western Australian Branch, B.M.A.: Branch.
MAR. 18.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 23.—New South Wales Branch, B.M.A.: Council.
MAR. 24.—Victorian Branch, B.M.A.: Council.
MAR. 25.—South Australian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi to xviii.

BALMAIN AND DISTRICT HOSPITAL, BALMAIN, NEW SOUTH WALES: Junior Resident Medical Officer.
COLLINSVILLE DISTRICT HOSPITAL, COLLINSVILLE, QUEENSLAND: Medical Officer.
COMMONWEALTH OF AUSTRALIA, DEPARTMENT OF HEALTH: Medical Officer.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Relieving Assistant Gynæcological Surgeon.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.
UNIVERSITY OF CAMBRIDGE, ENGLAND: Diploma in Medical Radiology and Electrology.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
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VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
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